The DECAF Score: Prognostication Scoring System for Patients Hospitalised with Acute Exacerbation of Chronic Obstructive Pulmonary Disease.

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CERTIFICATE

This is to certify that the dissertation titled **"The DECAF Score: Prognostication Scoring System for Patients Hospitalised with Acute Exacerbation of Chronic Obstructive Pulmonary Disease."** is the bonafide original work of Dr. Saranya S in partial fulfillment of the requirements for M.D. Branch – XVII (Tuberculosis and Respiratory Diseases) Examination of the Tamilnadu DR. M.G.R Medical University to be held in April 2015.

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DECLARATION

I, Dr.Saranya S solemnly declare that the dissertation titled "**The DECAF Score: Prognostication Scoring System for Patients Hospitalised with Acute Exacerbation of Chronic Obstructive Pulmonary Disease**" is a bonafide work done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 from March 2013 to September 2014 under the guidance and supervision of my Director Prof. Dr. D.Ranganathan, MD, Head of Department, Institute of Thoracic Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

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CONTENTS

ABSTRACT

The DECAF Score : Prognostication Scoring System for Patients Hospitalised with Acute Exacerbations of Chronic Obstructive Pulmonary Disease

Introduction :

Chronic obstructive pulmonary disease (COPD) is the fourth most frequent cause of death. In patients getting admitted with acute exacerbation of COPD (AECOPD), identifying simple, immediately accessible and strong prognostic indicators will aid in management decision.

Aim of the study :

To assess the DECAF score as an optimal clinical tool for accurate In-hospital prognostication of patients admitted with acute exacerbation of chronic obstructive pulmonary disease.

Materials and Methods :

90 patients admitted with primary diagnosis of AECOPD were included. Patients were scored according to the DECAF scoring system – Dyspnea, Eosinopenia, Consolidation, Acidemia and atrial Fibrillation. The patients were

followed during the entire hospital stay. The clinical outcome was categorized as a)improved b) status quo c) mortality. The role of DECAF score in predicting in-hospital outcome was analysed.

Results :

Out of 90 patients studied, 44 patients had DECAF score between 0-1 (low risk), 15 patients had a DECAF score of 2 (Intermediate risk) and 31 patients had a DECAF score between 3-6 (high risk). In the high risk group (DECAF 3-6) there was higher mortality, longer hospital stay and increased need for use of ventilator.

Conclusion :

The DECAF score incorporates indices routinely available and helps to stratify patients admitted with AECOPD into clinically relevant risk groups. It aids the physician in taking management decisions.

Key words:

Acute Exacerbation of Chronic Obstructive Pulmonary Disease, DECAF score, Prognosis.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable disease that is characterised by persistent airflow limitation which is usually progressive and associated with enhanced chronic inflammatory response in the airways and lung to noxious particles or gases. Exacerbations and comorbidities contribute to overall severity in patients[1]. COPD is the fourth most frequent cause of death after Ischemic heart disease, Cerebrovascular disease and Malignancy. COPD is a common cause of morbidity and mortality worldwide. The disease leads to huge economic and social burden which seems to be increasing day by day. The Global Burden of Disease Study projected that COPD, which is ranked sixth as a cause of death in 1990, will become the third leading cause of death in 2020[2]. This increasing mortality is mainly due to increasing trend of smoking, reduced mortality from other common diseases and increasing longevity of world population.

Acute Exacerbation of COPD (AECOPD) is an acute event characterised by worsening of patient's symptoms that is beyond normal day to day variations and leads to a change in medication. Exacerbations accelerate the rate of decline of lung function and are associated with significantly high mortality[3]. Another commonly used definition of AECOPD was given by Anthonisen and colleagues[4]. Three cardinal symptoms have to be present in order to define an episode as acute

exacerbation: increased sputum quantity, altered sputum quality(increased purulence), and increased dyspnea. According to these criteria, an exacerbation can be classified into three types

TABLE 1 : Types of AECOPD

Type I (most severe)	**Type II**	**Type III**
All three symptoms (i.e. increased sputum volume, increased sputum purulence and increased dyspnea).	Any two symptoms present	One symptom present plus at least one of the following: • An upper respiratory tract infection in the past 5 days • Increased wheezing • Increased cough • Fever without an obvious source • A 20% increase in respiratory rate • Heart rate above baseline

Exacerbations have a negative impact on patient's quality of life, affect symptoms and lung function taking several weeks to recover from. In patients presenting with hypercapnic exacerbations, the in-hospital death rate is around 10%[5]. If a patient is put on mechanical support during hospitalisation the death rate reaches 40% at one year after discharge[6]. The mortality three years after discharge reaches 49%[7]. The burden of COPD can be tackled by a comprehensive approach which

includes prevention, prompt diagnosis and immediate management of exacerbations. COPD exacerbations are precipitated by respiratory tract infections[8], air pollution[9], congestive heart failure, pulmonary embolism and interruption of maintenance therapy. The cause for about one third of exacerbations cannot be identified. When the number of exacerbations per year is two or more, then the COPD patient is called a "frequent exacerbator" [10]. Acute exacerbations, have a negative impact on the natural course of the disease. Roughly one to four decompensation episodes occur in a year in a COPD patient. Such exacerbations negatively influence the quality of life. These episodes lead to great healthcare and financial burdens. In a COPD patient 10 out of 100 times medical admissions are for an exacerbation episode and around 2% of all emergency department visits are due to exacerbations. Almost 60% of the economic burden of the disease is related to exacerbation episodes, especially severe acute exacerbations needing hospitalisation[11].

Diagnosis of acute exacerbation of Chronic Obstructive Pulmonary Disease is mainly based on clinical presentation of increasing dyspnea, increasing quantity and change in quality of sputum. A panel of biomarkers are yet to be identified for diagnosing an exacerbation. Similarly sufficient clinical data does not exist to determine the adequate duration of hospitalisation in these patients. Multiple prognostic indices related to higher death rates in COPD like Forced Expiratory Volume in

one second[12], Patient age[13], Hypoxemia[14], Hypercapnia[15], Comorbidity, Pulmonary hypertension, Body mass Index[16,17], have been investigated. Studies assessing prognostic factors in AECOPD patients who are hospitalised have been performed infrequently. Robust clinical tools which aid in management decisions have not been developed. Well established scores like BODE[18] score exist to assess mortality risk in stable COPD. Prognostic tools derived for stable state disease have not been studied on patients requiring hospitalisation. Prognostic research in exacerbations needing hospitalisation has been limited. There seems to be considerable difference in prognostic factors in acute exacerbation and stable COPD. There is need for identifying simple, easy to use, easy to obtain but strong predictors of in-hospital mortality. These predictive factors should also help in deciding the need for post-hospital care.

J Steer et al [19] developed a simple prognostication tool in acute exacerbation of COPD – the DECAF score, that will help in deciding location of care, early stepping up of care and anticipation of need for ventilatory support. It helps the physician in informing the relatives and patients on prognosis and risks associated with exacerbations. Thus it will help in directing the most efficient use of resources and thereby reducing mortality and morbidity.

REVIEW OF LITERATURE

Dyspnea severity and Mortality:

In COPD, the extent of breathlessness is assessed using the Medical Research Council Dyspnea(MRCD) Score. Compared to Forced Expiratory Volume in one second (FEV1), MRCD score for dyspnea appears to be a better predictor of mortality[20]. Its predictive value in exacerbations needing hospital admissions has not been adequately studied. However there are few reports to show that higher MRCD scores are predictors of both long and short term mortality. It has been shown that greater 'functional dependence' independently predicts hospital readmission and performance status. An important predictor of three month mortality following hospitalisation is a patient's inability to manage self care. In those patients who survive upto discharge, long term mortality is higher if there is higher level of functional dependence. Thus severe disability strongly influences the management considerations of individual patients. When dyspnea assessment is combined with a measure of functional dependence, predictive ability of the traditional dyspnea scoring scale can be improved.

J Steer et al[21], in a retrospective study described an improved version of MRCD scale – the extended MRC Dyspnea score(eMRCD), which was better in identifying patients at risk of repeated hospitalisation when compared to the MRCD scale though the latter is more frequently used. They studied a large population of patients with acute exacerbation

of COPD. The ability of both MRCD and eMRCD scores in predicting in-hospital death and early admission were compared.

Table – 2 Extended Medical Research Council Dyspnea Score

Limitation due to breathlessness	**MRCD**	**eMRCD**
Breathless only with strenuous exercise	1	
Breathless when hurrying on level / walking up a slight hill	2	
Walks slower than peers, or stops when walking on the flat at own pace	3	
Stops after walking 100m, or for a few minutes on the level	4	
Too breathless to leave the house	5	.
& independent in washing and / or dressing	**.**	**5a**
& dependent in washing and dressing	**.**	**5b**

In their study with 920 participants, 96 patients died in the hospital. The mortality rate for eMRCD 5a was 17.3% (30/96). The mortality rate for eMRCD 5b was 33.1% (47/96) (p=0.0012). In the non pneumonic AECOPD, the in-hospital death rates for patients with eMRCD 5b was significantly more than those with eMRCD 5a with p=0.048. In patients with pneumonic AECOPD , there was similar association. However it was not statistically significant (p=0.069). The eMRCD 5b group had higher 28 day readmission rates compared to the eMRCD 5a group (p=0.044). The prognostic ability of MRCD, eMRCD, and CURB-65 in assessing short term mortality were compared using areas under

receiving operator characteristics curve. In the study population, the in-hospital mortality was better predicted by eMRCD when compared to MRCD (p=0.0012) and CURB-65(0.019). In the non pneumonic AECOPD group, eMRCD had better discrimination compared to both MRCD (p=0.057) and CURB-65 (p=0.053), though it was statistically not significant. In pneumonic exacerbation, eMRCD scored significantly better than CURB-65. This study shows that when the traditional MRCD scale is extended to take into account a person's functional dependence (eMRCD), the predictive strength of the grading system is improved. eMRCD is better at assessing both in-hospital mortality and subsequent need for readmission following discharge. This grading system helps to classify a subgroup of patients who are at higher risk for in-hospital mortality (33.1% with eMRCD 5b). Assessing the of severity of dyspnea in patients requiring hospitalisation for AECOPD is easy to perform. Dyspnea grade is a potent predictor of outcome and provides important information which could aid in management decisions.

Mortality and Eosinopenia.

There are many studies that have assessed eosinophil counts, especially eosinopenia, as an indicator of infection, inflammation and bacteremia. The sample size in these studies is quite small and they have included heterogeneous populations. This may have led to contradictory results, thereby forming an important limitation for their interpretation.

In 2003 Gil et al[22], studied the role of eosinophil count in patients with infection. They showed that presence of elevated total leukocyte count (more than 10,000/mm^3) along with low eosinophil count(less than 40/mm^3) was significantly associated with the occurence of bacterial infections. Subsequently, Abidi et al[23], studied eosinopenia as a marker of sepsis. They concluded that low eosinophil count may be used as an indicator of infection in routine medical practice.

In a cohort[24] of 2,311 patients with bacteremia, investigators found that when compared with normal eosinophil count, eosinopenia (<50/mm^3) was associated with a 4.77-fold increase in mortality. When confounding factors were removed, persistently below-normal eosinophil count was found to be an independent but strong predictor of mortality.

Abidi et al[23]. evaluated the role of eosinopenia in predicting in-hospital mortality. The study was done in patients admitted in Intensive Care , a large proportion of whom had infection. Eosinopenia was

strongly associated with mortality at 28 days. In the multivariate analysis, the hazard ratio was 1.8.

Holland et al[25] studied sixty six patients hospitalised with exacerbation of chronic obstructive pulmonary disease. Admission eosinophil count was obtained in all 66 patients. The mortality rate in patients with eosinopenia at baseline was 17.4%. The mortality rate in patients with normal baseline eosinophil count was 2.4%. Mortality in eosinopenia was significantly higher when compared to patients with normal eosinophil values at p=0.049. Similarly group with eosinopenia had significantly longer duration of hospital stay (8 vs 5 days p=0.005). These authors concluded that besides routinely used indicators, low eosinophil count could be a used as an independent marker of disease severity and prognosis. It has been shown in animal models that in the presence of acute infection or inflammations, the leukocytes are diverted towards the formation of polymorphonuclear cells thereby leading to a low eosinophil count. Thus eosinopenia occurs when the body responds to acute infection. This is not dependent on adrenal glucocorticosteroids. In patients requiring intensive care, eosinopenia is an independent and useful marker of sepsis. Low eosinophil count occurring in the setting of acute exacerbation of COPD may actually be reflective of the extent of accompanying inflammatory response.

Coexistent pneumonia in acute exacerbation of COPD:

When a known case of COPD develops typical symptoms of an exacerbation episode due to community acquired pneumonia, there is always a doubt in the clinician's mind which it is right to call such an episode as AECOPD. Exacerbations in COPD are frequently associated with radiographic consolidation. Doubts have always existed as to whether patients with AECOPD and coexistent consolidation be actually diagnosed as AECOPD, with varying practices world wide. However patients with concurrent pneumonia were not excluded from major national studies of COPD exacerbation and non invasive ventilation in the United Kingdom. In these studies, concurrent pneumonic consolidation occurred in 16% and 34.2% respectively[26,27]. Further a conventional chest radiography may not be highly sensitive in identifying parenchymal consolidation. It has been seen that in quite a few patients who had an initially negative radiography, subsequent more detailed evaluation revealed the presence of consolidation[28].

Patients with pneumonic exacerbations of COPD had the same socio-demographic profile and severity of underlying disease when compared to subjects with non-pneumonic exacerbations. However the former group had more severe clinical and physiological derangement[29]. In both pneumonic and non-pneumonic exacerbations, the severity of airway obstruction and pathogens involved are similar. Exacerbations

associated with pneumonia should not be treated as just pneumonia, but need proper treatment of the AECOPD. Hence these patients will need continuous low flow oxygen, parenteral steroids, nebulised bronchodilators. If hypercapnic respiratory failure occurs noninvasive ventilation should be given. This suggests that coexistent pneumonia helps to identify patients with a higher severity of acute illness. It does not signify a different disease process[30].

Pneumonic and Non pneumonic acute exacerbations of COPD

David Lieberman et al[31] conducted a study in tertiary care Medical Centre in South Israel. Twenty three hospitalisations for pneumonic acute exacerbation of COPD (PNAE) and 217 hospitalisations for non pneumonic exacerbation of COPD (NPAE) were included. Patients with community acquired pneumonia were also included in the diagnosis of AECOPD due to following reasons. –

1. Patients had clinical features which are consistent with accepted criteria for AECOPD. The clinician gets to know the occurrence of pneumonia only by radiographic evaluation. It is not rational to eliminate the diagnosis of acute exacerbation of COPD in such patients. It is more apt to say that these patients have pneumonic exacerbation. This strengthens the importance of combination of events.

2. Most of the patients with AECOPD are managed on an out-patient setup. In such a scenario, chest radiographs are not routinely obtained. Hence elimination of patients with community acquired pneumonia is not practically possible. They showed that compared to NPAE, patients with pneumonia had higher rates of hypoxemia, higher rates of hospitalisation(P=0.004), higher rates of sudden onset (P=0.005). Patients with PNAE also had higher rates of ICU admission (P=0.006), intubation(p=0.01), in-hospital death (P=0.007) and longer duration of stay in hospital(P=0.001). In PNAE, Viral and pneumococcal etiologies are more common.

Table3 - Comparison of Hospital Factors Between Pneumonic AECOPD and Nonpneumonic AECOPD Groups

Variable	Pneumonic AECOPD (n=23)	Non pneumonic AECOPD (n=217)	P value
Invasive ventilation	4(17)	10(5)	0.01
Admission to ICU	6(26)	14(7)	0.006
Mortality	3(13)	2(1)	0.007
Hospitalization,days	7.9[8.3]	4.6[4.1]	0.001
Readmission	2(10)	36(17)	NS
Recovery within 30 days	16(80)	166(77)	NS

Data are presented as No.(%) or No.[SD].

Table 4 - Comparison of the frequency distribution of infectious etiologies between pneumonic AECOPD and non pneumonic AECOPD Groups

Etiology	Pneumonic AECOPD (n=23)	Nonpneumonic AECOPD (n=217)	P Value
Viral agents			
Influenza virus type A	0(0)	23(11)	NS
Influenza virus type B	3(13)	12(6)	NS
Parainfluenza virus type 1	3(13)	16(7)	NS
Parainfluenza virus type 2	9(39)	29(13)	0.004
Parainfluenza virus type 3	1(4)	6(3)	NS
Adenovirus	5(22)	15(7)	0.03
Respiratory syncytial virus	2(9)	14(7)	NS
At least one of the above	18(78)	99(46)	0.003
Bacterial agents			
S pneumoniae	10(43)	38(18)	0.006
H influenzae	3(13)	7(3)	NS
M catarrhalis	1(4)	8(4)	NS
At least one of the above	10(43)	48(22)	0.02
Atypical bacterial agents			
Legionella spp.	5(22)	35(16)	NS
M pneumonia	3(13)	31(14)	NS
At least one of the above	8(35)	64(30)	NS
Agent not identified	1(4)	64(30)	0.001

Data are presented as No.(%).

J Steer et al[21] showed that co-existent pneumonia is common in patients with acute exacerbation of COPD. It is also associated with high mortality rate. When there is simultaneous occurrence of AECOPD and pneumonia, the mortality rates are higher than pneumonia alone. A total of 920 were patients included in the study. Pneumonic AECOPD patients had longer hospital stay (seven days) compared to patients with non pneumonic AECOPD (six days, $p<0.001$). The in-hospital death

rates for non pneumonic AECOPD(36/621) was 5.8% whereas 20.1% with PNAE died in hospital(60/299). 28 day readmission rates for pneumonic AECOPD was 19.5% and non pneumonic AECOPD was 18%. There was no statistical difference in readmission rates (P=0.62).

Table 5 – pAECOPD and In-hospital mortality

pAECOPD and CURB-65	In hospital mortal ity
0-1	11%
2	16%
3-5	31.2%

CURB-65 is often used to stratify patients with pAECOPD. It is frequently used to guide treatment in these patients. However it is clear from the data that CURB-65 is not an accurate predictor of risk of mortality in this population. When statistically analysed it is seen that CURB-65 performs moderately with AUROC = 0.661. eMRCD with AUROC = 0.759, performed better in predicting short term (p=0.017) as well as post hospital follow up moratlity (p=0.040). Recent studies have shown that CURB-65 is a good predictor of in-hospital mortality in non pneumonic AECOPD. From the above study it is clear that eMRCD has out-performed CURB-65 for all patients.

Acidemia and mortality :

The frequency of hypercapnic respiratory failure in patients with AECOPD varies from 16-35% with overall mortality of 35-43%[32]. Hypoxia was a common condition on hospital admission as well as hypercapnia. Respiratory acidosis (arterial pH $\geq$ 7.35 and/or PCO2 $\leq$ 6.0kPa, 45 mm hg) is an indication for ventilator support in AECOPD[1]. It can be provided either by noninvasive (by nasal or facial mask) or invasive ventilation (by oro-tracheal tube or tracheostomy). Mechanical ventilation decreases acute respiratory acidosis. It reduces tachypnea, work of breathing, severity of dyspnea and duration of stay in hospital .

Karin H. Groenewegen et al[33] studied a total of 171 patients admitted with AECOPD. The in-hospital death rate was 8%. The death rate at 1 year of follow up was 23%. The in-hospital mortality rate for patients requiring intensive care management was comparable at 6%. However in patients admitted to the ICU for respiratory failure, the 1-year follow up mortality rate was significantly higher at 35%. The multivariate Cox proportional hazards model was used to determine independent predictors of survival. Variables included in the regression model were age, sex, FEV_1, $_PO_2$, $_PCO_2$, body mass index, long-term use of oral corticosteroids, comorbidity index, and hospital readmissions. The maintenance use of oral glucocorticosteroids (relative risk [RR], 5.07;

95% confidence interval [CI], 2.03 to 12.64), $_{P}CO_2$ (RR, 1.17; 95% CI, 1.01 to 1.38), and age (RR, 1.07; 95% CI, 1.01 to 1.12) were independently related to mortality. They showed that when the characteristics of ICU patients and non-ICU patients were compared, ICU patients had higher $_{P}CO_2$ and lower pH values.

Table 6 - Characteristics of Patients Transferred to ICU

Characteristics	ICU Patients (n=17)	Non-ICU Patients (n=154)	p Value
Age, yr	73.4±6.5	70.4±8.6	NS
FEV_1,% predicted	34.2±12.2	39.1±15.4	NS
PaO_2, kPa	8.0±5.0	7.4±2.7	NS
PaO_2, kPa	9.3±2.7	6.5±1.9	0.0001
$_{P}H$	7.31±0.9	7.39±0.7	0.0001
BMI,kg/m^2	26.1±6.5	24.1±4.2	NS
Comorbidity index	1.50±0.89	1.55±0.90	NS
Corticosteroid use, No.	2	15	NS
Length of stay, days	9.45	16.88	0.005

Values given as mean±SD, unless otherwise indicated. NS=not significant.

Chronic alveolar hypoventilation leads to hypercapnia. Thus hypercapnia is reflective of severity of the respiratory disease. Hence compared to patients with normoventilation, those with persistently

elevated pCO_2 have poor prognosis. Patients with chronic hypercapnia constituted a major chunk of the study group.

In one study[34], patients with severe COPD with at least one hospital admission for hypercapnic respiratory failure were compared to patients who had been treated for unresectable non-small cell lung cancer. The results of this study showed that COPD patients had significantly less ability to perform the activities of daily life, lower physical, social and emotional functioning than did patients with non-small cell lung cancer. This study confirms that COPD patients do not receive the necessary palliative care that is needed.

Steer et al[19] showed in their study that arterial pH was statistically lower in patients who died in hospital compared to those who survived till discharge (pH <7.3 odds ratio (95% CI) 2.68(1.41 – 5.09) p=0.003).

Atrial fibrillation and COPD:

COPD is associated with high risk of cardiac arrhythmias. Hypoxemia[35], acidosis[36], corpulmonale[37], coexisting ischemic heart disease[38] have been considered major causes of arrhythmias in COPD. The type and risk of arrhythmias occurring in a COPD patient is determined by severity of the disease. Supraventricular tachycardias are the most common arrhythmias occuring during exacerbations . However, even in patients with stable COPD the incidence of cardiac arrhythmias is

considerable. Commonly atrial fibrillation (AF) is seen in patients hospitalised for exacerbations. AF is by far the most common arrhythmia in the elderly population.

P.Buch et al[39] analysed data from 13,430 males and females. The participants were taken from the Copenhagen City Heart Study. None of the subjects had previous myocardial infarction. Re-examination was done after 5 years to look for new arrythmias. Multivariate analyses were used with adjustment for cardiopulmonary risk factors. At the time of hospitalisation 290 cases of AF were diagnosed (2.20%). There were 62 new cases of AF at 5-yr follow-up (0.58%). Risk of new AF at re-examination was 1.8-times higher for FEV1 between 60–80% of predicted compared with FEV1 > 80% after adjustment for sex, age, smoking, blood pressure, diabetes and body mass index. The risk of hospitalisation for AF was 1.3-times higher for FEV1 between 60–80% and 1.8-times higher for FEV1<60% compared with FEV1 ≥80%, when additional adjustment was made for education, treatment with diuretics and chest pain at activity. The authors concluded that reduced lung function is an independent predictor for incident atrial fibrillation.

Table 7.Presence of atrial fibrillation (AF) at baseline, at re-examination and at incident hospitalizations according to lung function

	FEV1% predicted		
	<60%	60-80%	□80%
At baseline Subjects n	1259	3904	8351
Presence of AF	15(1.20)	32(0.82)	31(0.37)
At re-examination Subjects n	809	2947	6910
Presence of AF	9(1.11)	25(0.85)	28(0.41)
At hospital admission Subjects n	1167	3699	8315
All cases of AF	47(4.03)	96(2.60)	147(1.77)
AF as main diagnosis	46(3.94)	28(0.76)	19(0.23)

Data are presented as n(%) unless otherwise stated. FEV_1 predicted : forced expiratory volume in one second % predicted.

The mechanism connecting reduced lung function with AF is not clear. Recent observations[40] have revealed that ectopic beats initiating AF often originate in the walls of the pulmonary veins. It is possible that this could be triggered by changes in gas composition or pulmonary hypertension. Hypoxia and corpulmonale could only account for some of this effect since the relationship was also found in subjects with mild to moderately reduced FEV1. Reduced lung function has been shown to be an independent predictor of IHD and of stroke, and it is possible that the biological mechanism for development of AF could be linked to

atherosclerosis via a common pathway of development of vascular and airways disease, e.g. foetal or early childhood exposure[41].

Predictors of outcome in acute exacerbation of COPD:

Roche et al[30] studied COPD patients visiting emergency department due to exacerbation. They identified simple, accessible but strong predictors of in-hospital mortality and the need for post hospital support. They found 3 simple clinical criteria that were important predictors of in-hospital death.

The criteria were

1. Age
2. Clinical severity at entry
3. Baseline dyspnea grade

Factors considered in clinical severity were

1. Cyanosis,
2. Lower limb edema,
3. Asterixis,
4. Neurological impairment,
5. Use of inspiratory accessory muscles,
6. Expiratory use of abdominal muscles.

The following table presents the results of Multivariate logistic regression analysis:

TABLE - 8 : Odds ratio for death and need for post-hospital support at discharge on multivariate logistic regression analysis

	Risk of death	Post-hospital support need
Females versus males	4.5(1.6-12.1)	2.2(1.4-3.4)
Age≥70yrs		3.4(2.1-5.5)
Clinical signs of severity at entry		
Cyanosis	1.5(0.7-3.0)	1.6(1.0-2.6)
Neurological impairment	5.1(2.4-10.8)	3.3(1.6-6.7)
Lower limb oedema	1.0(0.4-2.0)	1.0(0.5-1.5)
Asterixis	1.7(0.6-4.3)	0.7(0.3-1.7)
Use of inspiratory accessory muscles	2.6(1.1-6.2)	1.6(1.0-2.7)
Expiratory use of abdominal muscles	0.9(0.4-1.9)	1.2(0.7-2.0)
Baseline dyspnea grade		
0-1	1.0	1.0
2-3	3.6(0.7-16.5)	1.3(0.7-2.2)
4-5	6.5(1.4-29.3)	2.0(1.1-3.6)

Data are presented as odds ratio (95% confidence interval) when all clinical signs of severity are individually integrated into the model.

In 2001, The American College of Physicians and The American Society of Internal Medicine conducted an evidence based process[42]. Only 11 studies had been conducted to identify predictors of in-hospital mortality. In the 5 largest studies with multivariate analysis (N=322-3050), the independent predictors of in-hospital deaths were

1. Age
2. Acute physiology score

3. Body mass Index
4. Functional status before exacerbation
5. $_{P}O_2$/Inspiratory oxygen fraction ratio.
6. Need of mechanical ventilation
7. Serum Albumin
8. Sodium levels
9. Cardiac comorbid conditions

None of these studies provided a simple prediction tool to aid in management decisions.

Yan Chang et al[43] conducted a study to assess all cause death in COPD. Cross-sectional study of patients with the discharge diagnosis of COPD, utilizing the Premier Perspective database was carried out. Patients aged 40 years and above were selected if they had a primary discharge diagnosis of COPD. All data analyses were based on individual level. Predictors for mortality were identified by multiple logistic regressions. Bonferroni correction for multiple logistic regression models was adapted to control family-wise errors. After excluding outliers, the bivariate logistic regressions were conducted between mortality and the independent variables. From the results of the univariate analyses, the mortality risk of patients of COPD increased by 4% for every increase in age by 1 year and female patients were 19% less likely to die compared to male patients. The highest risk of mortality was in the patients who were

Asian (odds ratio [OR] = 1.65; confidence interval [CI] = 1.02-2.65), had insurance (OR=3.31; CI=2.63-4.16), were admitted with elective admission (OR=1.95; CI=1.67-2.27), had extreme severity of illness (OR=38.26; CI=29.61-49.43), had extreme risk of mortality (OR=88.56; CI=67.97-115.39), and were assigned with Deyo-adapted Charlson Index score of 4 and above(OR=2.98; CI=2.50-3.55). Elixhauser comorbidities including valvular disease(OR=1.50; CI=1.07-2.10), other chronic lung disease (OR=7.62; CI=4.53-12.80), renal failure (OR=6.39; CI=3.03-13.46), metastatic cancer(OR=3.24; CI=2.45-4.27), solid tumor without metastasis (OR=2.10; CI=1.66-2.67), and weight loss (OR=5.26; CI=4.34-6.37) were more likely to happen to patients who died than who survived; however, comorbid conditions such as hypothyroidism (OR=0.82; CI=0.61-0.98) and depression (OR=0.68; CI=0.54-0.85) were associated with decreased risk of morality. Oral/parenteral corticosteroids (OR=0.76; CI=0.66-0.88) and antibiotics (OR=0.65; CI=0.56-0.75) had Odds Ratio less than 1.0, which indicated that they had a protective effect on mortality.

John Steer et al[19], developed a robust clinical prediction tool. He studied a large population of COPD patients getting hospitalised with exacerbations. 920 patients from diverse geographic locales were recruited. Socio-demographic and clinical profile collected. They aimed at developing a simple but easily usable prognostic tool. The strongest

five categorical variables selected and relative weights assigned according to regression co-efficient. Thus Dyspnea, Eosinopenia, Consolidation, Acidemia and Atrial fibrillation- The DECAF score was developed. DECAF score performed better for prediction of in-hospital mortality than other predictive instruments in AECOPD like Acute Physiology And Chronic Health Evaluation II prognostic index, the COPD and Asthma Physiology Score and BAP 65 score. The area under DECAF score ROC curve for predicting in hospital mortality was 0.86 (95% CI– 0.82 to 0.89)

AIMS AND OBJECTIVES

Aims of the study:

To assess the DECAF score as an optimal clinical tool for accurate In-hospital prognostication of patients admitted with Acute Exacerbation of Chronic Obstructive Pulmonary Disease.

METHODOLOGY

Subject selection:

Patients getting admitted to Rajiv Gandhi Government General Hospital (RGGGH) with symptoms of acute exacerbation of Chronic Obstructive Pulmonary Disease (COPD) were selected.

A patient was diagnosed to have AECOPD if

1. Age was above 35 years and
2. History of exposure to risk factors
    - Smoking history of >10 cigarette pack
    - Smoke from home cooking and heating fuels
    - Occupational dusts and chemicals and
3. Spirometric evidence of airflow obstruction (forced expiratory volume in one second (FEV1)/forced vital capacity(FVC) <0.70 without significant reversibility) when clinically stable within last two years PLUS presence of any one of the following
    - worsening of dyspnea above normal day to day variations
    - increased quantity of sputum production
    - increased purulency of sputum.

Inclusion criteria

- Patients admitted with primary diagnosis of acute exacerbation of chronic obstructive pulmonary disease
- Age ≥ 35 years

Exclusion criteria

1. Patients in whom the primary reason for admission was other than acute exacerbation of COPD were excluded from the study. Hence patients with the following diseases were excluded from our study

- Bronchial Asthma-acute exacerbation
- Bronchiectasis-infective exacerbation
- Interstitial Lung Diseases-exacerbation
- Lung cancer
- Pneumothorax
- Congestive cardiac failure
- Acute on chronic decompensated liver disease
- Acute on chronic decompensated renal disease
- Psychiatric illness

All of these exclusion criteria were left to the clinician's discretion in order to ensure that the real life nature of the study was respected.

2. Previous inclusion in the study.

Study centres

The study was conducted at a premiere tertiary care institute - Rajiv Gandhi Government General Hospital, Park Town, Chennai

Study design

- The study was a prospective study.

- No specific intervention was carried out.
- Consecutive patients admitted with the diagnosis of acute exacerbation of COPD during the study period were included in the study . No specific method of randomisation was used.
- No controls were used in the study

Study period:

18 months March 2013 – September 2014

Data collection

The following were assessed in our study in patients with Acute Exacerbation Chronic Obstructive Pulmonary Disease (COPD)

- Socio-demographic and clinical data
- Details of comorbidity
- Complete blood count and absolute eosinophil count at admission
- Arterial blood gas results at admission
- Chest radiograph
- Electrocardiogram

Methodology

90 consecutive patients admitted with the diagnosis of acute exacerbation of COPD satisfying our inclusion and exclusion criteria during the study period were included .Socio-demographic and clinical data of the study subjects were collected on admission. Breathlessness

was graded according to the extended Medical Research Council (MRC) dyspnea (eMRCD) Score[21]. This subdivides patients too breathless to leave the house unaided (traditional MRCD 5) into those who are able to independently manage washing and/or dressing (eMRCD 5a) and those and those requiring assistance with both (eMRCD 5b). Details of comorbidity were obtained from the clinical notes. Cardiac assessment was done by cardiologists. Complete blood count, absolute eosinophil count and arterial blood gas results performed at the time of admission were recorded. Chest radiograph was assessed by the treating physician to look for new consolidation. The presence of atrial fibrillation was confirmed by ECG at the time of hospital admission. The patients were followed during the entire hospital stay. Treatment was individualised for each patient. The investigator did not interfere with the treatment. Patients were scored according to the DECAF scoring system[1], wherein the following parameters are given points,

Table – 9: The DECAF Score

Variable	Score
Dyspnea eMRCD 5a eMRCD 5b	 1 2
Eosinopenia (<50cells/mm3)	1
Consolidation	1
Acidemia (pH <7.3)	1
Atrial fibrillation	1
Total score	6

DECAF: Dyspnea according to eMRCD, extended MRC dyspnea, Eosinopenia, Consolidation, Acidemia and atrial Fibrillation;.

The clinical outcome was categorised as

a) improved

b) status quo

c) mortality.

"Improved" is clinically defined as subjective sense of improvement and objective improvement in dyspnea scoring. "Status quo" refers to patients who get discharged against medical advice and whose clinical condition at the time of discharge does not fit into the other two groups. The results were statistically analysed.

Extended Medical Research Council grades (eMRC)

Dyspnea is a complex subjective sensation. Quantification of dyspnea is a difficult task. However it is necessary to do so for research purposes. Fletcher and his colleagues developed a short questionnaire while studying the pulmonary symptoms of Welsh coal miners at the Medical Research Council Pneumoconiosis Unit in the early 1940s. The questionnaire allowed the placement of a numeric value on the exercise capacity of each member of the study population. The questions were published in 1952. It was later developed into the MRC breathlessness scale[47]. The MRC breathlessness grades do not quantify breathlessness like the Borg scale or visual analogue scales. Instead, it quantifies the disability associated with breathlessness by identifying when breathlessness occurs or by quantifying the associated exercise impairment. There is a strong agreement between observers recording MRC dyspnea grades. The grades correlate well with other dyspnoea scales, lung function measurements and with objective measures of disability such as six minute walking distance. J Steer et al[21] expanded the traditional MRC Score to develop extended Medical Research Council (MRC) dyspnea (eMRCD) Score, this subdivides patients too breathless to leave the house unaided (traditional MRCD 5) into those who can independently manage washing and/or dressing (eMRCD 5a) and those requiring assistance with both (eMRCD 5b). The eMRCD Score is more

strongly associated with inhospital mortality than the traditional score. The patients were asked to choose the description that best suited their condition.

Absolute eosinophil count:

Hingleman's solution stains the eosinophils , lyses the red cells and other leukocytes. 0.33ml of diluting fluid is mixed with 0.04ml of blood and kept for 10 minutes. The counting chamber is charged. Fuchs Rosenthal chamber with a depth of 0.2mm and a ruled area of 16 sq mm used. The cells in all 16 squares are counted under low power objective with 10X eyepiece.

$$\text{Absolute eosinophil count} = \frac{\text{no. of cells counted x dilution}}{\text{area counted x depth.}}$$

Eosinopenia is defined as absolute eosinophil count < 50/microlitre.

Arterial blood gas analysis:

For arterial blood gas analysis ABL80 FLEX analyser was used. ABL80 FLEX analyzer consists of the analyzer, a multi-use disposable sensor cassette and a solution pack. 2ml blood from radial artery or femoral artery is collected in ABG syringe. The tip of the inlet probe is fully immersed in the sample. The ABL80 FLEX CO-OX analyzer with OSM software configuration aspirates a sample volume of approximately

65 µL for each measurement. This volume is automatically aspirated into the analyzer during the sample analysis procedure. The results are displayed after the analysis.

Electrocardiogram:

The presence of atrial fibrillation on ECG was diagnosed with the help of following findings

- Irregularly irregular rhythm.
- Absence of P waves.
- Absence of an isoelectric baseline.
- Varying ventricular rate.
- QRS complexes usually < 120 ms unless pre-existing bundle branch block, accessory pathway, or rate related aberrant conduction.
- Fibrillatory waves may be present and can be either fine (amplitude < 0.5mm) or coarse (amplitude >0.5mm).

Pulmonary Function Testing :

Patients who had spirometric evidence of airway obstruction (% FEV_1<70%) without reversibility were included in the study. Spirometry is a physiological test that measures how an individual inhales or exhales

volumes of air as a function of time. Spirometry is done as per the American Thoracic Society recommendations. The appropriate technique of spirometry has to be demonstrated to each patient individually before the start of procedure. The patients are asked to inhale rapidly and completely upto Functional Residual Capacity (FRC). The patients are instructed to hold the mouth piece in their mouth, sealed tightly by their lips. Care has to be taken to prevent occlusion by tongue. Patients are asked to blast out air without any hesitation and are asked to completely exhale. Throughout the procedure, patients should be coached using body languages and phrases. The testing has to be done in sitting position and nose clips are used. Acceptability and repeatability criteria as recommended by ATS[48] are

WITHIN-MANOEUVRE CRITERIA

Individual spirograms are “acceptable”if

They are free from artefacts

- Cough during the first second of exhalation
- Glottis closure that influences the measurement
- Eary termination or cut-off
- Effort that is not maximal throughout
- Leak
- Obstructed mouthpiece

They have good starts

- Extrapolated volume <5% of FVC or 0.15 L, whichever is greater

They show satisfactory exhalation

- Duration of ≥ 6 s (3 s for children) or a plateau in the volume-time curve or if the subject cannot or should not continue to exhale.

Between-manoeuvre criteria

After three acceptable programs have been obtained, apply the following tests.

The two largest values of FVC must be within 0.150 L of each other

The two largest values of FEV_1 must be within 0.150 L of each other

If both of these criteria are met, the test session may be concluded

If both of these criteria are not met, continue testing until

Both of the criteria are met with analysis of additional acceptable spirograms or

A total of eight tests have been performed (optional) or

The patient/subject cannot or should not continue

Save, as a minimum, the three satisfactory manoeuvres.

The test is then repeated after administration of salbutamol through nebulisation to see for reversibility

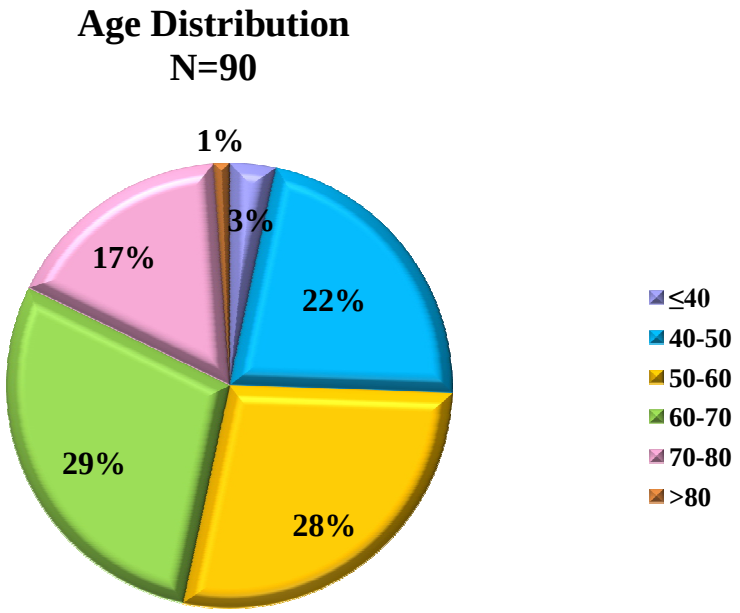
Statistical analysis

Statistical analysis was done using the SPSS softwares . Significance of correlation between variables was assessed using p value. A correlation was considered to be statistically significant if its p value was less than 0.05.

RESULTS

Age distribution:

A total of 90 patients were included in our study as per our patient selection methods, inclusion and exclusion criteria. The age group of our patients in our study ranged from 37 to 82. The mean age of the study population was 59.6 with a standard Deviation of 10.6. The number of patients in the age groups ≤40, 40-50, 50-60, 60-70, 70-80, >80 were 3 (3.3%), 20 (22.2%), 25 (27.8 %), 26 (28.9%), 15(16.7%) and 1(1.1%) respectively.

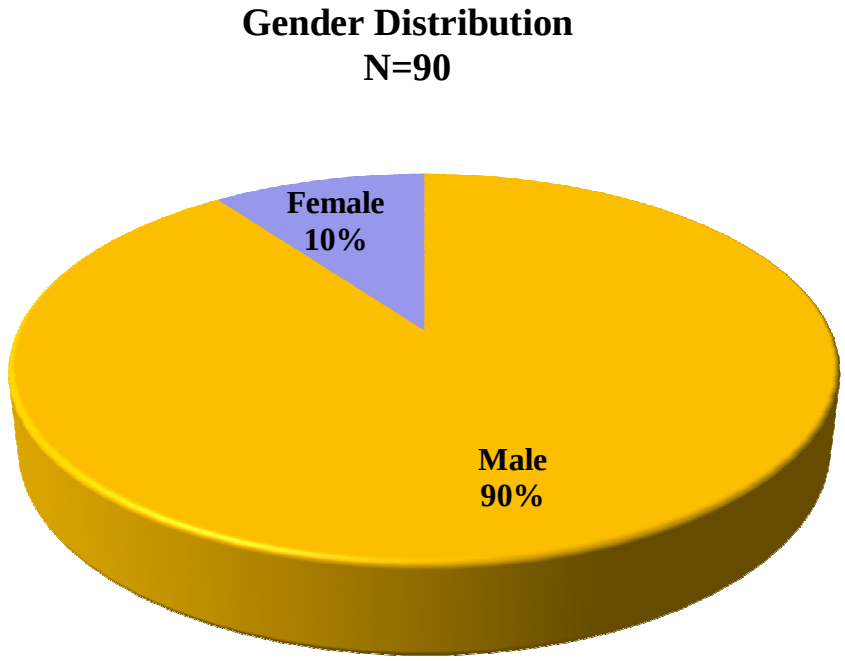


Age	≤40	40-50	50-60	60-70	70-80	>80
No	3	20	25	26	15	1

Fig 1: Age distribution

Gender distribution:

Out of the 90 patients in the study, 81 are male and 9 are female. Thus males accounted for 90% of our study population while females accounted for 10%.

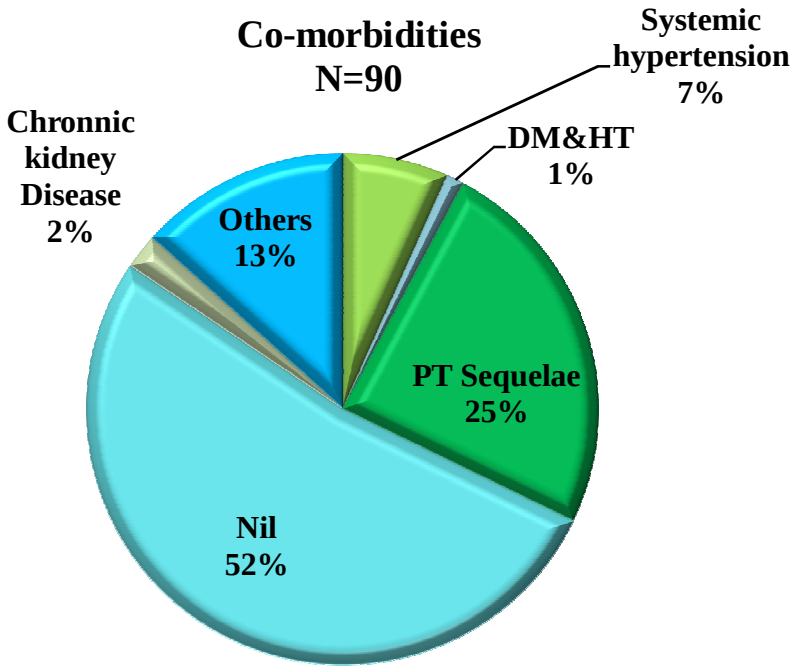


Gender	Male	Female
No	81	9

Fig 2: Gender Distribution

Comorbidity:

In the study population, 47 did not have any comorbid illness. The most common comorbidity among the study population is Pulmonary Tuberculosis Sequalae. Among 'others' 4 patients had coronary artery disease, 2 patients had obstructive sleep apnea, 3 patients had connective tissue disorders, 3 patients had hypersensitivity pneumonitis

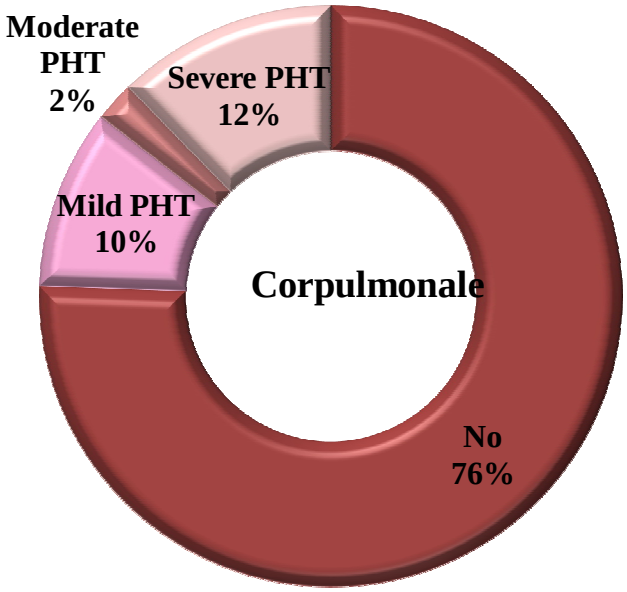


Comorbidity	No
Systemic hypertension	6
Diabetes mellitus and Systemic hypertension	1
PTB sequalae	22
Chronic kidney disease	2
Other	12
Nil	47
Total	90

Fig 3 : Comorbidities

Presence of cor pulmonale:

Out of 90 patients included in the study 22 (24.4%) patients had corpulmonale as evidenced on echo. Out of them 9 (10%) patients had mild pulmonary hypertension (PHT), 2 (2.2%) patients had moderate PHT, 11 (12.2%) patients had severe PHT.

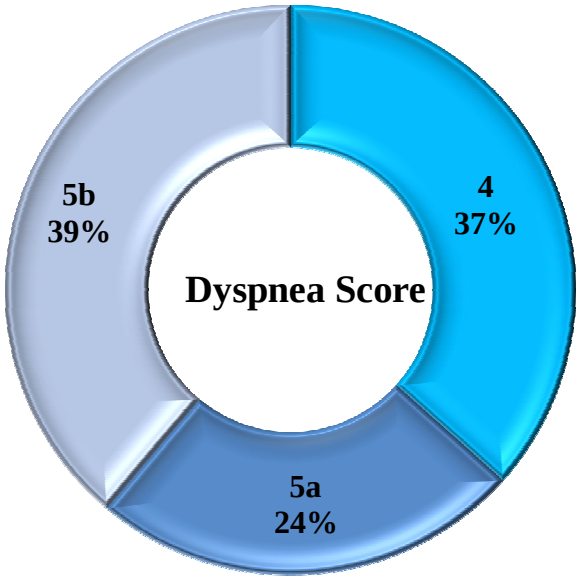


Corpulmonale	No
No	68
Mild PHT	9
Moderate PHT	2
Severe PHT	11
Total	90

Fig 4 : Corpulmonale

Dyspnea grading:

The patients in the study were graded according to the extended Medical Research Council score. Accordingly, 33 patients had eMRC grade 4, 22 patients had eMRC grade 5a and 35 patients had a score of 5b. In terms of percentage, the distribution of patients in grades 4, 5a and 5b was 36.7, 24.4 and 38.9 respectively.

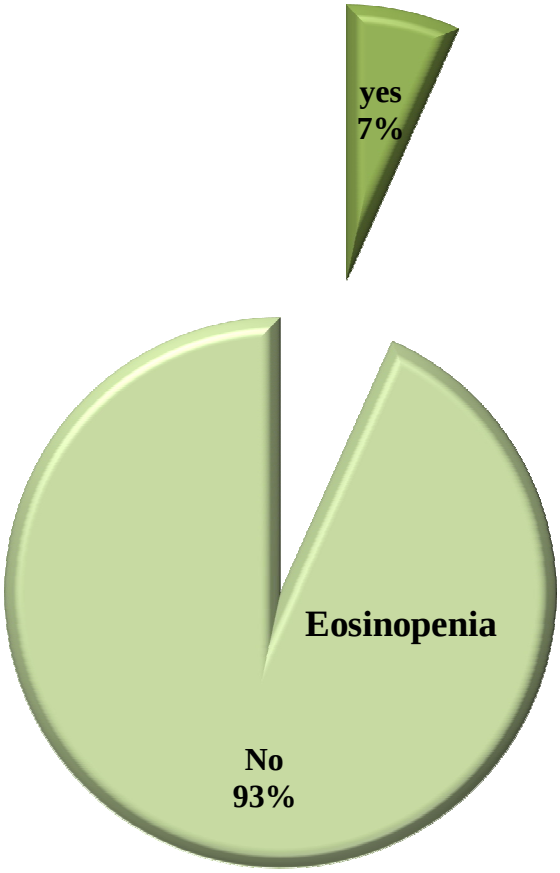


Dyspnea grade eMRC	No
4	33
5a	22
5b	35

Fig 5 : Dyspnea Score

Presence of Eosinopenia:

Eosinopenia was defined as an absolute eosinophil count of less than 50/mm3. 6 out of 90 patients had eosinopenia. Hence 6.7% of the study population had low eosinophil count.

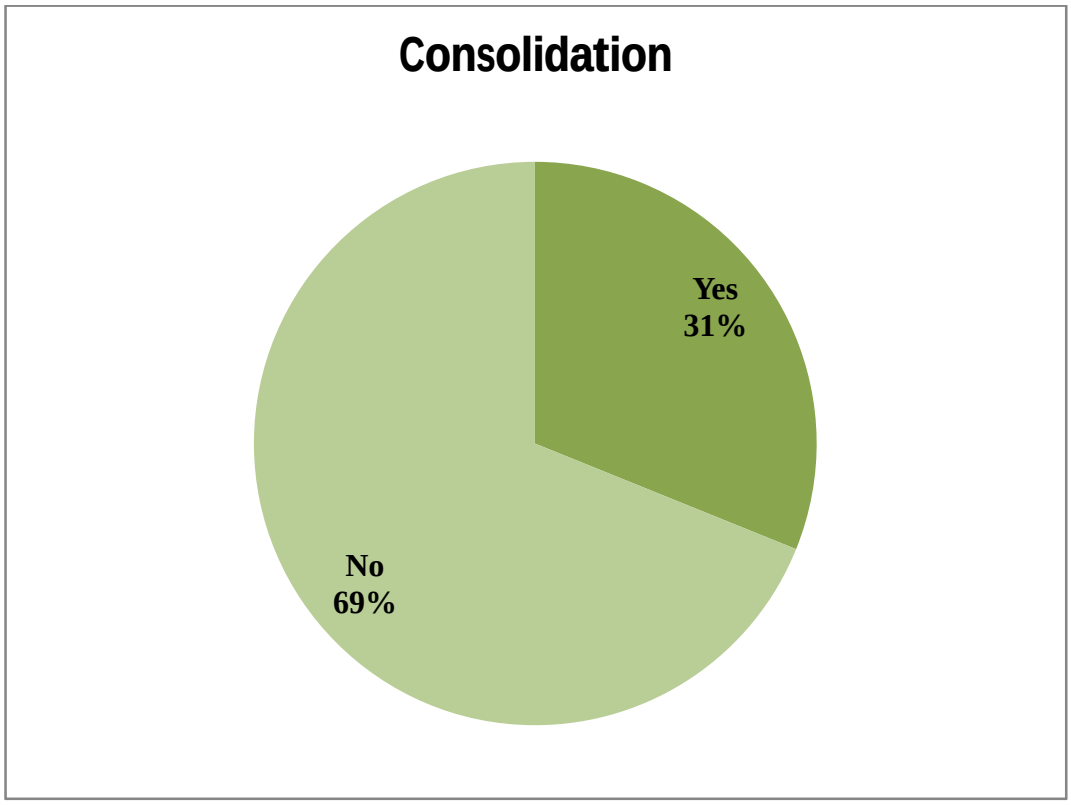


Eosinopenia	No
Yes	6
No	84

Fig 6 : Eosinopenia

Presence of Consolidation:

Assessment of chest radiographs of patients at admission to confirm the presence of consolidation was done. Accordingly 28 (31%) patients had consolidation on chest radiograph.

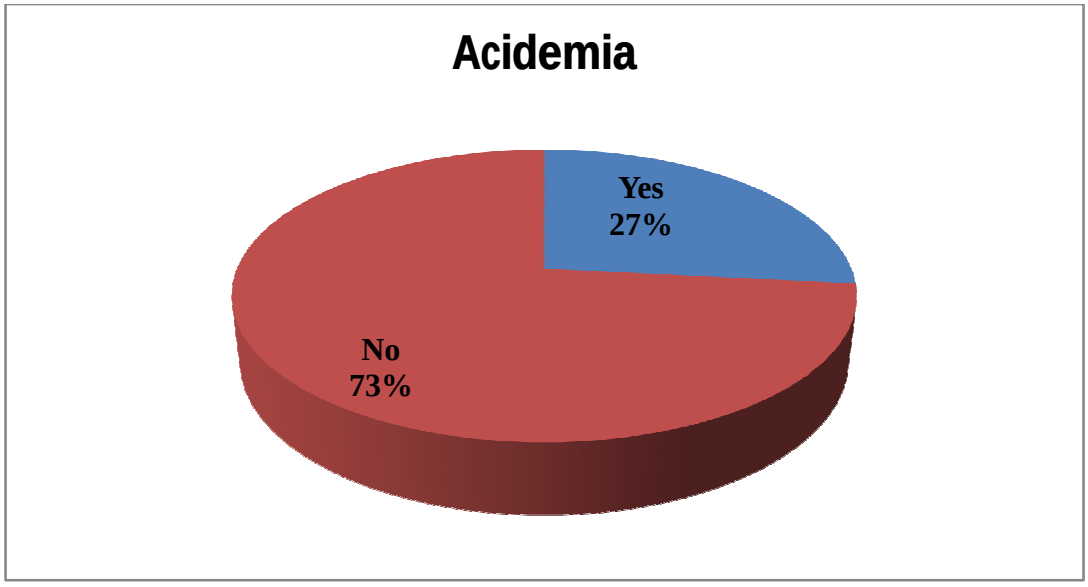


Consolidation	No
Yes	28
No	62

Fig 7 : Consolidation

Presence of Acidemia:

Acidemia is defined as the presence of arterial blood gas pH<7.30. 24 patients (26.6%) had acidemia.

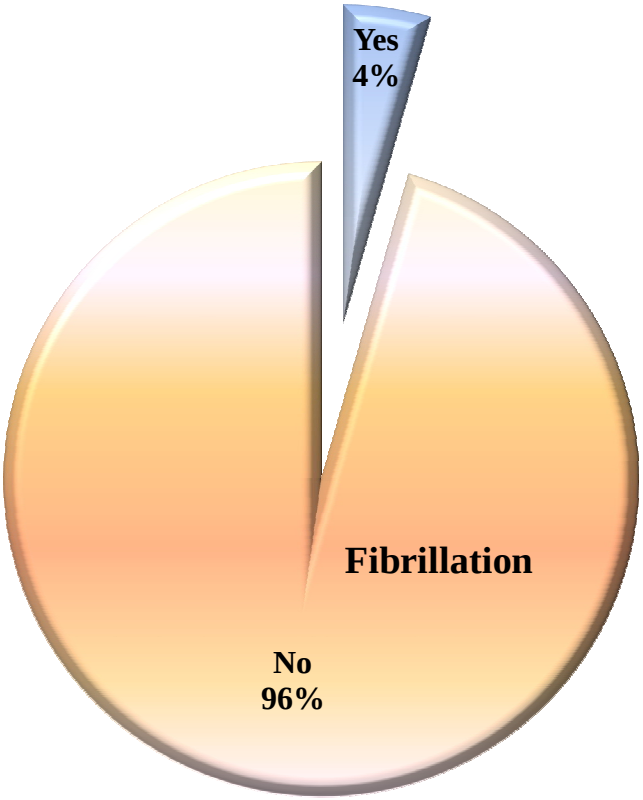


Acidemia	No
Yes	24
No	66

Fig 8 : Acidemia

Presence of Fibrillation:

Presence of atrial fibrillation was confirmed with the presence of admission electrocardiogram. Accordingly 4 (4.4%) patients had atrial fibrillation, while the remaining 86 did not have fibrillation.



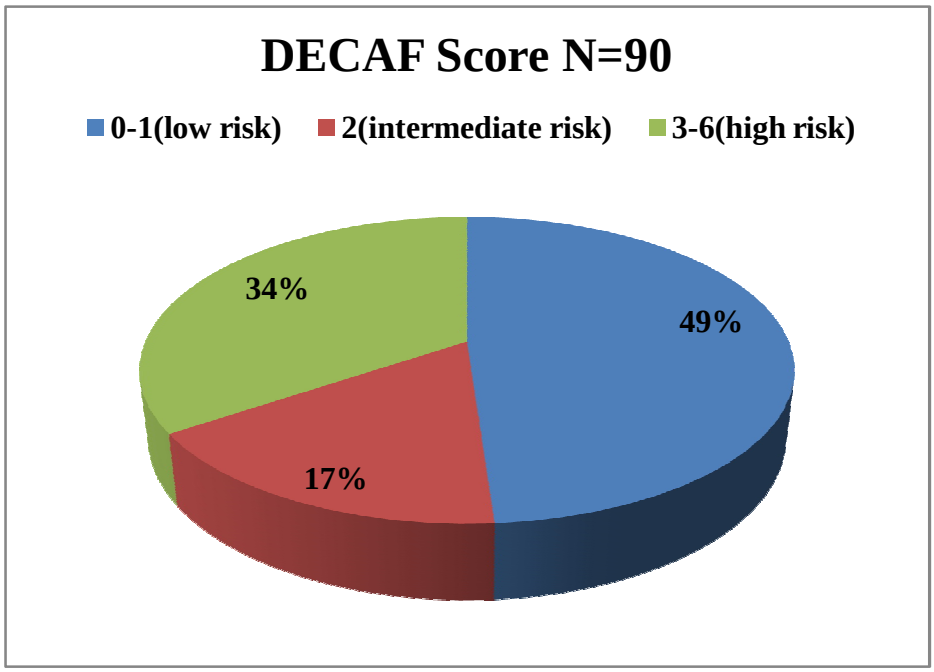
Atrial Fibrillation	No
Yes	4
No	86

Fig 9 : Atrial Fibrillation

THE DECAF SCORE:

Each patient was scored using DECAF score – where dyspnea eMRC grade 5a gets 1 point, dyspnea eMRC grade 5b gets 2 points, others parameters, namely Eosinopenia, Consolidation, Acidemia, atrial Fibrillation get 1 point each. We divided the population into three groups namely low risk, intermediate risk and high risk with the groups getting DECAF score of 0-1, 2 and 3-6 respectively.

44 patients had a DECAF score between 0-1, 15 patients had a DECAF score of 2 and 31 patients had a DECAF score between 0-6. In terms of percentage this is 48.9%, 16.7% and 34.4% respectively.

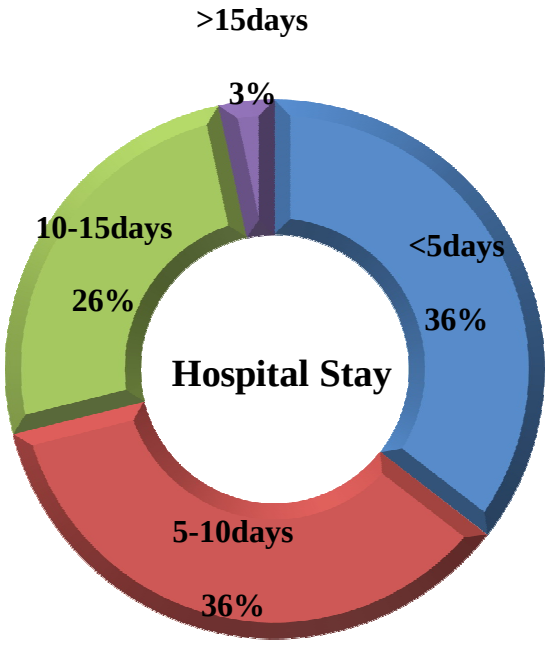


DECAF score	No
0-1(low risk)	44
2(intermediate risk)	15
3-6(high risk)	31

Fig 10 : DECAF score

Duration of hospital stay :

The average duration of hospital stay for the study group was 7 days. The length of hospital stay was divided into four groups- <5 days, 5-10 days, 10-15 days, >15 days. 32 (35.6%) patients had hospital stay lesser than 5 days, 32 (35.6%)patients had hospital stay between 5-10 days, 23(25.6%) patients had hospital stay between 10-15 days, 3 (3.3%) patients stayed in hospital longer than 15 days

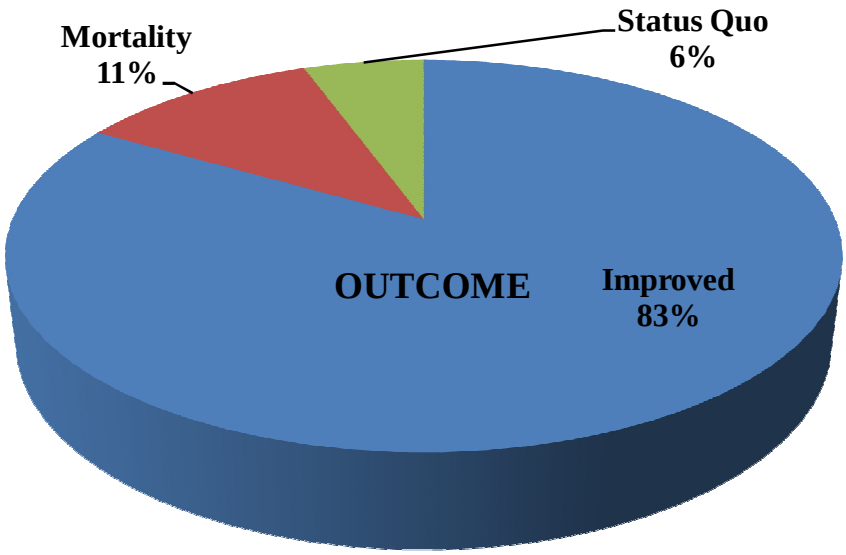


Hospital stay	No
<5days	32
5-10 days	32
10-15 days	23
>15 days	3

Fig 11 : Hospital Stay

Outcome:

The mortality rate for the study population was 10 out of 90 (11%). 75 patients “improved” at the time of discharge with “improved” being clinically defined as subjective sense of improvement and objective improvement in dyspnea scoring, 10 patients died in the hospital, 5 patients were discharged against medical advice whose clinical condition could not be defined as ‘improved’ or deteriorated at the time of leaving the hospital.

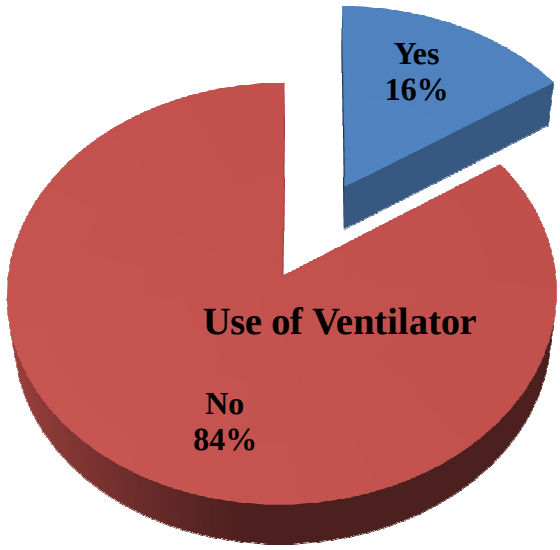


DECAF score	No
Improved	75
Mortality	10
Status Quo	5

Fig 12 : Outcome

Use of ventilator:

Out of the 90 patients 14(15.6%) were put on ventilator, 6 were put on Non invasive ventilation and 8 were put on Invasive ventilation.

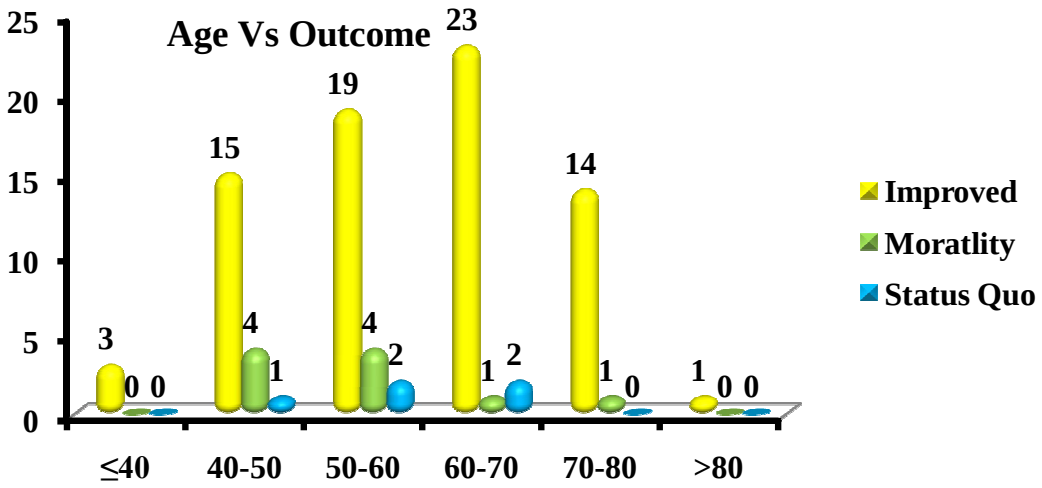


Use of ventilator	No
Yes	14
No	76

Fig 13 : Use of Ventilator

Age and outcome:

The mortality in the age groups 40-50, 50-60, 60-70,70-80 is 4,4,1,1 respectively. When age was taken as scattered variables, older patients showed higher mortality. However there is no statistically significant association between the age and outcome,P≥0.05(P=0.800).



Age Vs Outcome	Improved	Moratlity	Status Quo	Total
≤40	3	0	0	3
40-50	15	4	1	20
50-60	19	4	2	25
60-70	23	1	2	26
70-80	14	1	0	15
>80	1	0	0	1
Total	75	10	5	90

Fig 14 : Age Vs Outcome

Table 10 : Age * outcome Cross tabulation

			outcome			Total
			improved	status quo	mortality	
age	Below 40	Count	2	0	0	2
		Expected Count	1.7	.1	.2	2.0
		% within age_samples	100.0%	.0%	.0%	100.0%
		% within outcome_effect	2.7%	.0%	.0%	2.2%
	Age 41-50	Count	15	1	4	20
		Expected Count	16.7	1.1	2.2	20.0
		% within age_samples	75.0%	5.0%	20.0%	100.0%
		% within outcome_effect	20.0%	20.0%	40.0%	22.2%
	Age 51-60	Count	19	2	4	25
		Expected Count	20.8	1.4	2.8	25.0
		% within age_samples	76.0%	8.0%	16.0%	100.0%
		% within outcome_effect	25.3%	40.0%	40.0%	27.8%
	Age 61-70	Count	25	2	1	28
		Expected Count	23.3	1.6	3.1	28.0
		% within age_samples	89.3%	7.1%	3.6%	100.0%
		% within outcome_effect	33.3%	40.0%	10.0%	31.1%
	Age 70 and above	Count	14	0	1	15
		Expected Count	12.5	.8	1.7	15.0
		% within age_samples	93.3%	.0%	6.7%	100.0%

		% within outcome_effect	18.7%	.0%	10.0%	16.7%
Total		Count	75	5	10	90
		Expected Count	75.0	5.0	10.0	90.0
		% within age_samples	83.3%	5.6%	11.1%	100.0%
		% within outcome_effect	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	5.927[a]	8	.655
Likelihood Ratio	7.272	8	.508
Linear-by-Linear Association	2.473	1	.116

a. 11 cells (73.3%) have expected count less than 5. The minimum expected count is .11.

No Significant association is exist between Age and outcome.

Age and ventilator use:

The number of patients put on ventilator in the age groups 40-50, 50-60, 60-70 is 7, 6, 1 respectively. In the study group, the use of ventilator increases as the age advances. None of patients less than 40 years or above 70 years were put on ventilator. The use of ventilator is more with older patients. This relationship is statistically significant at p=0.020.

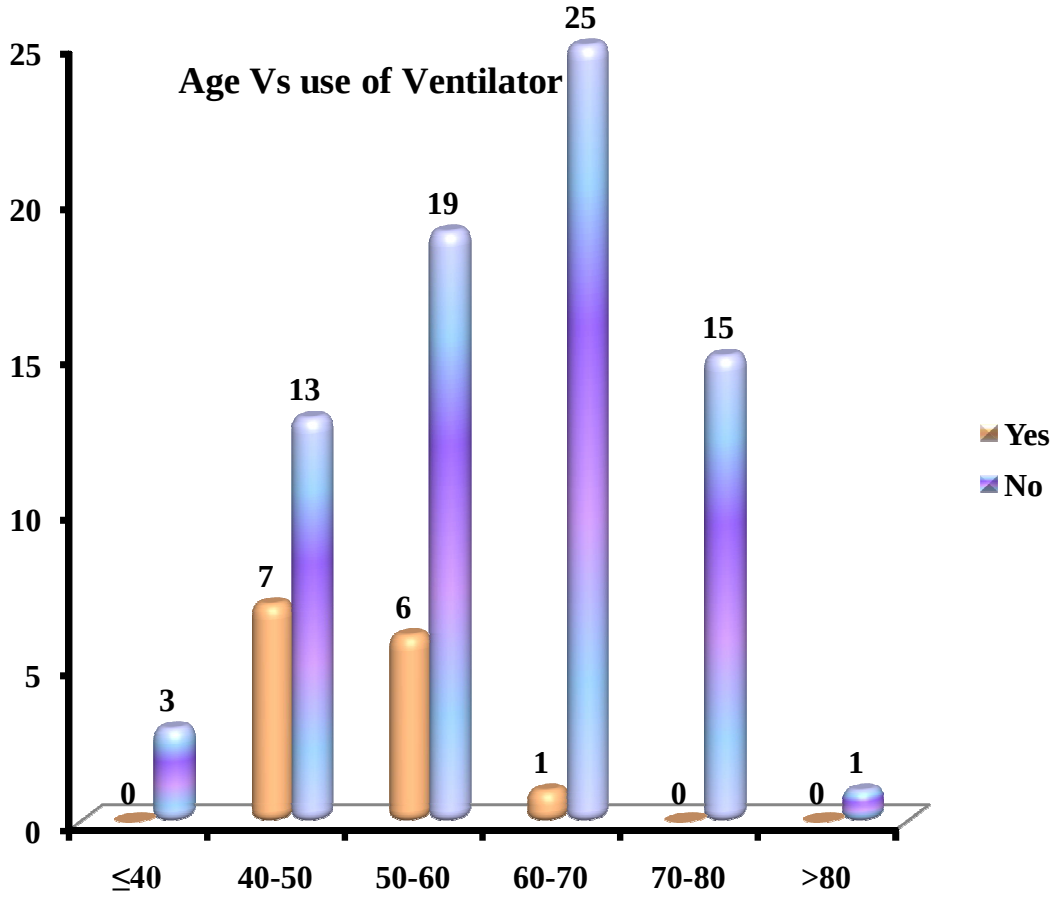


Fig 15 : Age with use of ventilator

Table 11 : Age Vs use of Ventilator

Age	Yes	No	Total	P-value
≤40	0	3	3	
40-50	7	13	20	
50-60	6	19	25	
60-70	1	25	26	**.020(S)**
70-80	0	15	15	
>80	0	1	1	
Total	14	76	90	

By chi-square test, there is a significant association between the age and the use of ventilator,P≤0.05(P=0.020).

Age and hospital stay:

In the study population the average number of hospital stay is 7 days. It is seen that as age advances the duration of hospital stay increases with older people having the need to stay longer. This association is statistically significant at p=0.055 (by ANOVA).

Table 12 : Age Vs Hospital Stay

Age	<5days	5 -10 days	10-15days	>15 days	Total	P-value
≤40	0	3	0	0	3	
40-50	3	9	7	1	20	
50-60	9	8	6	2	25	
60-70	13	7	6	0	26	**.055(S)**
70-80	6	6	3	0	15	
>80	1	0	0	0	1	
Total	32	33	22	3	90	

Influence of gender on outcome:

The mortality among female patients in the study is 2 out of 9 (18%). The moratlity among the male patients is 8 out of 68 (11.8%).There is no significant association between gender and outcome. This could be attributed to the very low number of female participants in the study.

Table 13 : Gender Vs Outcome

	outcome			Total
	Improved	**Mortality**	**Status Quo**	
Male	68	8.0	5.0	81.0
Female	7	2.0	0.0	9.0
Total	75.0	10.0	5.0	90.

Tabel 14 : Gender Vs outcome Crosstab

			sex		Total
			Male	Female	
out come_ effect	Improved	Count	68	7	75
		Expected Count	67.5	7.5	75.0
		% within outcome_effect	90.7%	9.3%	100.0%
		% within sex	84.0%	77.8%	83.3%
	status_ quo	Count	5	0	5
		Expected Count	4.5	.5	5.0
		% within outcome_effect	100.0%	.0%	100.0%
		% within sex	6.2%	.0%	5.6%
	Mortality	Count	8	2	10
		Expected Count	9.0	1.0	10.0
		% within outcome_effect	80.0%	20.0%	100.0%
		% within sex	9.9%	22.2%	11.1%
Total		Count	81	9	90
		Expected Count	81.0	9.0	90.0
		% within outcome_effect	90.0%	10.0%	100.0%
		% within sex	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.704[a]	2	.427
Likelihood Ratio	1.979	2	.372
Linear-by-Linear Association	.650	1	.420
N of Valid Cases	90		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .50.

There is no association between gender and outcome.

Impact of gender on duration of hospital stay and use of ventilator:

Male patients had longer in-hospital stay compared to female patients. This association is statistically significant at p=0.009. However there is no gender difference in the use of ventilator.

Table 15 : Gender and Hospital stay

Gender		Hospital stay				Total	P-value
		<5days	5 to 10 days	10 to 15 days	>15days		
	Male	28.8	28.8	20.7	2.7	81.0	
	Female	3.2	3.2	2.3	.3	9.0	**.009(S)**
Total		32.0	32.0	23.0	3.0	90.0	

By chi-square test, there is a significant association between the gender and hospital stay. $P \leq 0.05 (P=0.009)$.

Impact of gender on use of ventilator

12 out of 69 (17.4%) male patients were put ventilator whereas 2 out of 9 (22.2%) female patients were ventilated. There is no statistically significant gender difference in the need of ventilator use among the study population.

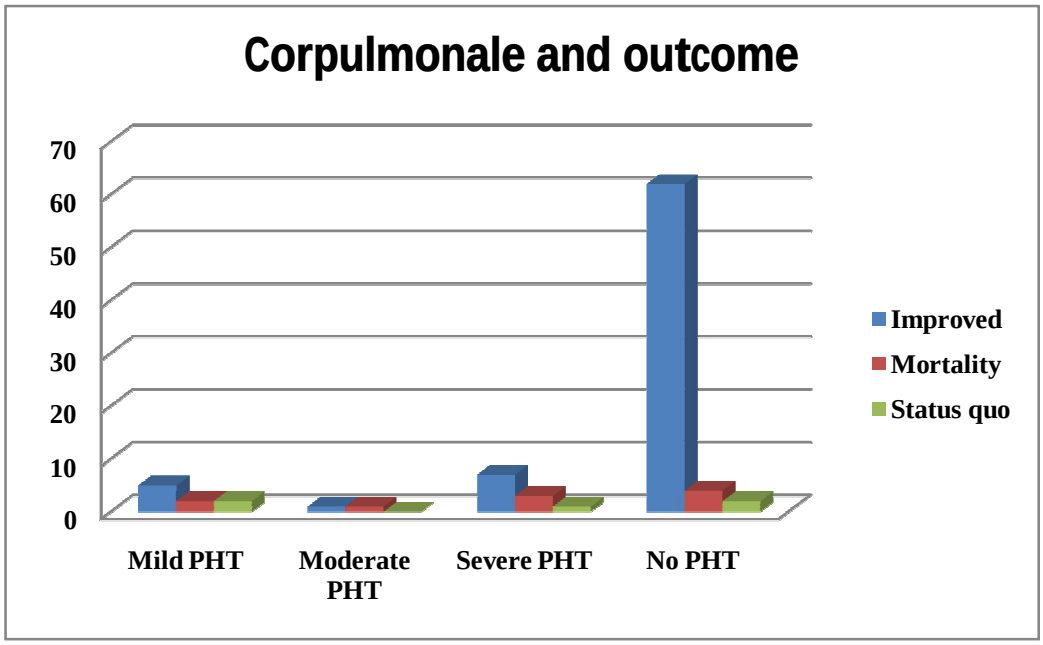
Table 16: Gender and use of ventilator

Gender		Use of ventilator		Total	P-value
		Yes	No		
	Male	12	69	81.0	
	Female	2	7	9.0	0.561(NS)
Total		14.0	76.0	90.0	

By chi-square tests, there is no significant association between the gender and use of ventilator, P≥0.05(P=0.561).

Impact of corpulmonale on outcome:

The mortality among patients with corpulmonale is 27.3 % (6/22). The mortality among patients without corpulmonale is 5.9% (4/68). Among the study population, patients having corpulmonale and pulmonary hypertension had higher mortality. Most of the patients who did not have PHT improved. This association between corpulmonale and outcome is statistically significant at p=0.015.



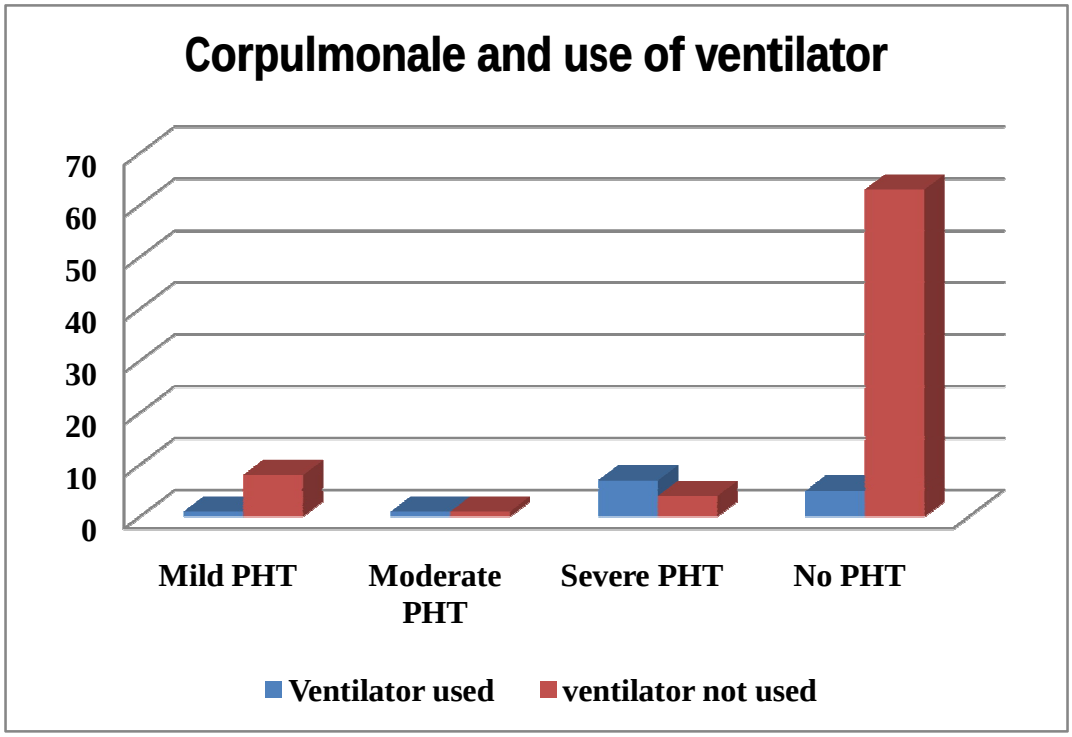
Corpulmonale	Outcome			Total	P-value
	Improved	Mortality	Status Quo		
Mild PHT	5	2	2	9.0	
Moderate PHT	1	1	0	2.0	**0.015(S)**
Severe PHT	7	3	1	11.0	
No PHT	62	4	2	68.0	
Total	75.0	10.0	5.0	90.0	

Fig 16 : Corpulmonale and outcome

By chi-square tests, there is a significant association between Cor pulmonale and outcome, P≤0.05, (P=.015).

Impact of corpulmonale on ventilator usage:

9 out of 22 (41%) patients with corpulmonale required ventilator support. 5 out of 63 (8%) persons without corpulmonale required ventilator support. There was increased need for usage of ventilators in patients with pulmonary hypertension compared to patients without PHT. This relationship was statistically significant at $p<0.05$. Most of the patients without PHT didn't require ventilator.



Corpulmonale	Use of ventilator		Total	P-value
	Yes	No		
Mild PHT	1	8	9.0	
Moderate PHT	1	1	2.0	**0.000(S)**
Severe PHT	7	4	11.0	
No PHT	5	63	68.0	
Total	14.0	76.0	90.0	

Fig 17 : Corpulmonale and use of ventilator

By chi-square test, there is a significant association between the Corpulomonale and use of ventilator, P=0.000.

Impact of corpulmonale on duration of in-hospital stay:

Patients with corpulmonale had a longer duration of hospitay stay. Patients without corpulmonale stayed for only 5-10 days. This relation was statistically significant at p =0.03.

Table 17 : Corpulmonale and hospital stay

Corpulmonale	Hospital stay				Total	P-value
	<5days	5 to 10days	10 to 15 days	>15days		
Mild PHT	3.2	3.2	2.3	.3	9.0	
Moderate PHT	.7	.7	.5	.1	2.0	**.003(NS)**
Severe PHT	3.9	3.9	2.8	.4	11.0	
No PHT	24.2	24.2	17.4	2.3	68.0	
Total	32.0	32.0	23.0	3.0	90.0	

By chi-square test, there is a significant association between the Corpulmonale and hospital stay, P≤0.05, (P=.003).

Impact of grade of dyspnea on outcome:

In patients getting admitted with AECOPD, as the eMRC dysnea grade increases, the mortality increases. Almost all the patients in the score of 4 and 5a got improved. Mortality is predominantly seen in eMRC 5b group. The mortality rate among eMRC 5b is 10 out of 35(28.6%). The relation is statistically significant at p=0.000.

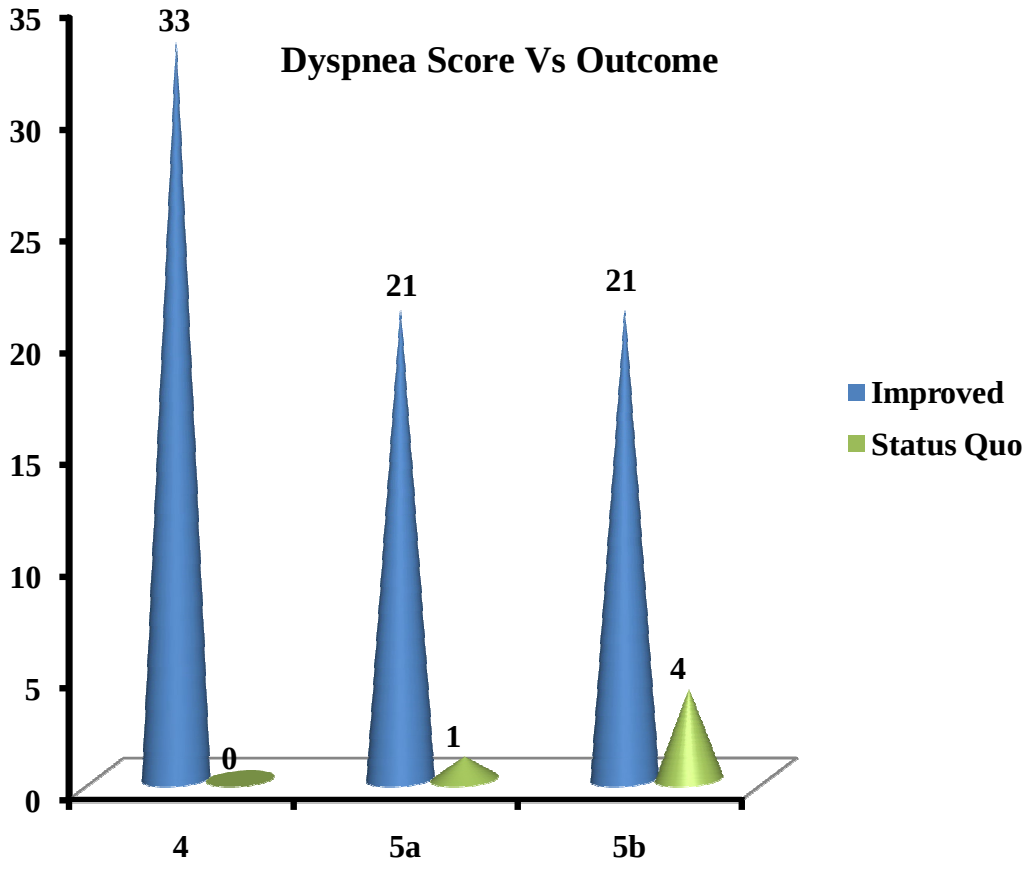


Fig 18 : Dyspnea Score Vs outcome

Table 18 : Dyspnea Score Vs outcome

Dysnea Score Vs Outcome	Improved	Moratlity	Status Quo	Total
4	33	0	0	33
5a	21	0	1	22
5b	21	10	4	35
Total	75	10	5	90

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	**23.536[a]**	**4**	**.000**
Likelihood Ratio	28.198	4	.000
Linear-by-Linear Association	18.916	1	.000
N of Valid Cases	90		

a. 6 cells (66.7%) have expected count less than 5. The minimum expected count is 1.22.

Significant association exists between dyspnea and outcome at p=0.000

Impact of dyspnea on duration of hospital stay:

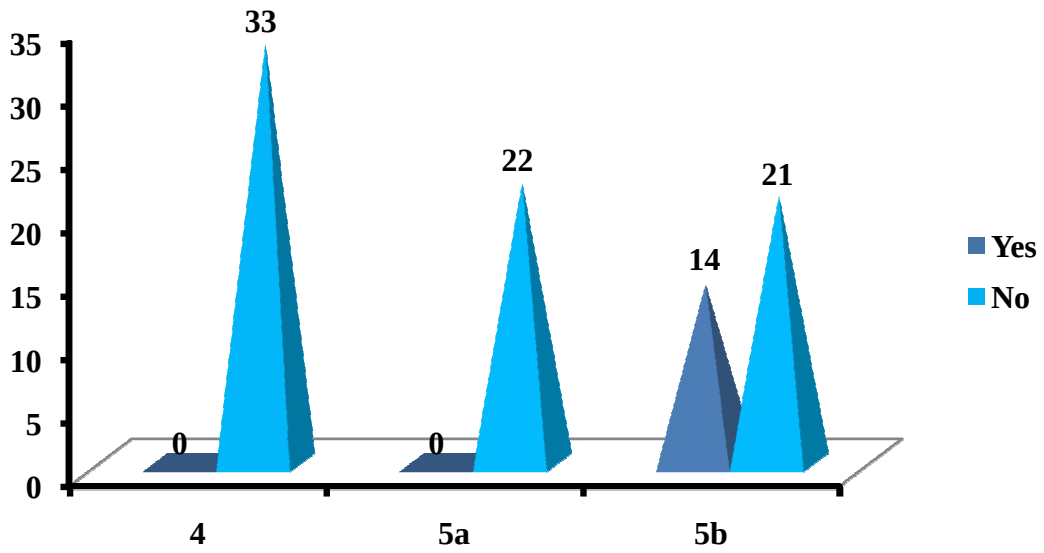
With increasing grade of dyspnea, the in-hospital stay for patients admitted with AECOPD increases . This association is statistically significant. By chi-square test, the association between the dysnea score and hospital stay is P=0.000 (P≤0.05).

Table 19 : Dyspnea score and hospital stay

Dysnea Score Vs Hospital Stay		Hospital stay				Total	P-value
		<5days	5 to10days	10 to 15 days	>15days		
	4	24	8	1	0	33.0	
	5a	4	11	7	0	22.0	**0.000(S)**
	5b	4	13	15	3	35.0	
Total		32.0	32.0	23.0	3.0	90.0	

Impact of dyspnea on ventilator use:

With increasing grade of dyspnea there is increased usage of ventilator. None of the patients in the score of 4 and 5a required ventilator whereas 66% in the dyspnea score of 5b required ventilator. This association is statistically significant at p=0.000.



Dyspnea Score Vs Use of ventilator	Yes	No	Total	P-value
4	0	33	33	**0.000(S)**
5a	0	22	22	
5b	14	21	35	
Total	14	76	90	

Fig 19 : Dyspnea and ventilator use

By chi-square test, there is a significant association between the dyspnea score and use of ventilator, P≤0.05,(P=0.000).

Impact of Eosinopenia on outcome:

All the 6 patients with eosinopenia improved. None of the patients with eosinopenia died during hospital stay. There is no significant association between eosinopenia and outcome.

Table 20 : Crosstab Eosinopenia and outcome

		Eosinopenia		Total
		yes	No	
outcome	improved	6	69	75
	status quo	0	5	5
	mortality	0	10	10
Total		6	84	90

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.286a	2	.526
Likelihood Ratio	2.272	2	.321
Linear-by-Linear Association	1.160	1	.281
N of Valid Cases	90		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .33.

No significant association between eosinopenia and outcome

Impact of Consolidation on Outcome:

9 out of the 10 (90%) patients in the mortality group had chest X-ray features of consolidation. Out of 75 patients who improved 16 (21.3%) had consolidation. Presence of consolidation is associated with higher mortality. This association is statistically significant at p=0.000

Table 21 : Crosstab consolidation and outcome

		consolidation		
		Yes	No	**Total**
Outcome	improved	16	59	75
	status_quo	4	1	5
	mortality	9	1	10
Total		29	61	90

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	**24.583[a]**	**2**	**.000**
Likelihood Ratio	23.879	2	.000
Linear-by-Linear Association	23.136	1	.000
N of Valid Cases	90		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.61.

Significant association exists between consolidation and outcome.

Impact of acidemia on outcome:

All 10 (100%) patients with in-hospital mortality had arterial blood pH<7.30. 14 out of 75(18.7%) patients who improved had acidemia. Presence of acidemia is associated with higher in-hospital mortality. This relation is statistically significant at p=0.00

Table 22 : Crosstab acidemia and outcome

		Acidemia		
		yes	**no**	**Total**
Outcome	Improved	14	61	75
	status quo	1	4	5
	Mortality	10	0	10
Total		25	65	90

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	**29.254[a]**	**2**	**.000**
Likelihood Ratio	29.145	2	.000
Linear-by-Linear Association	25.589	1	.000
N of Valid Cases	90		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.39.

Significant association exists between acidemia and outcome.

Impact of atrial fibrillation on outcome:

In the study group 2 patients had atrial fibrillation. Both the patients died during the course of treatment. In terms of percentage, 20% of the patients with in-hospital mortality had atrial fibrillation. Presence of atrial fibrillation is associated with higher mortality. By Chi square this relation is statistically significant at p=0.03.

Table 23 : Crosstab atrial fibrillation and outcome

		Fibrillation		**Total**
		yes	**no**	
Outcome	improved	2	73	75
	Status quo	0	5	5
	mortality	2	8	10
Total		4	86	90

Chi-Square Tests

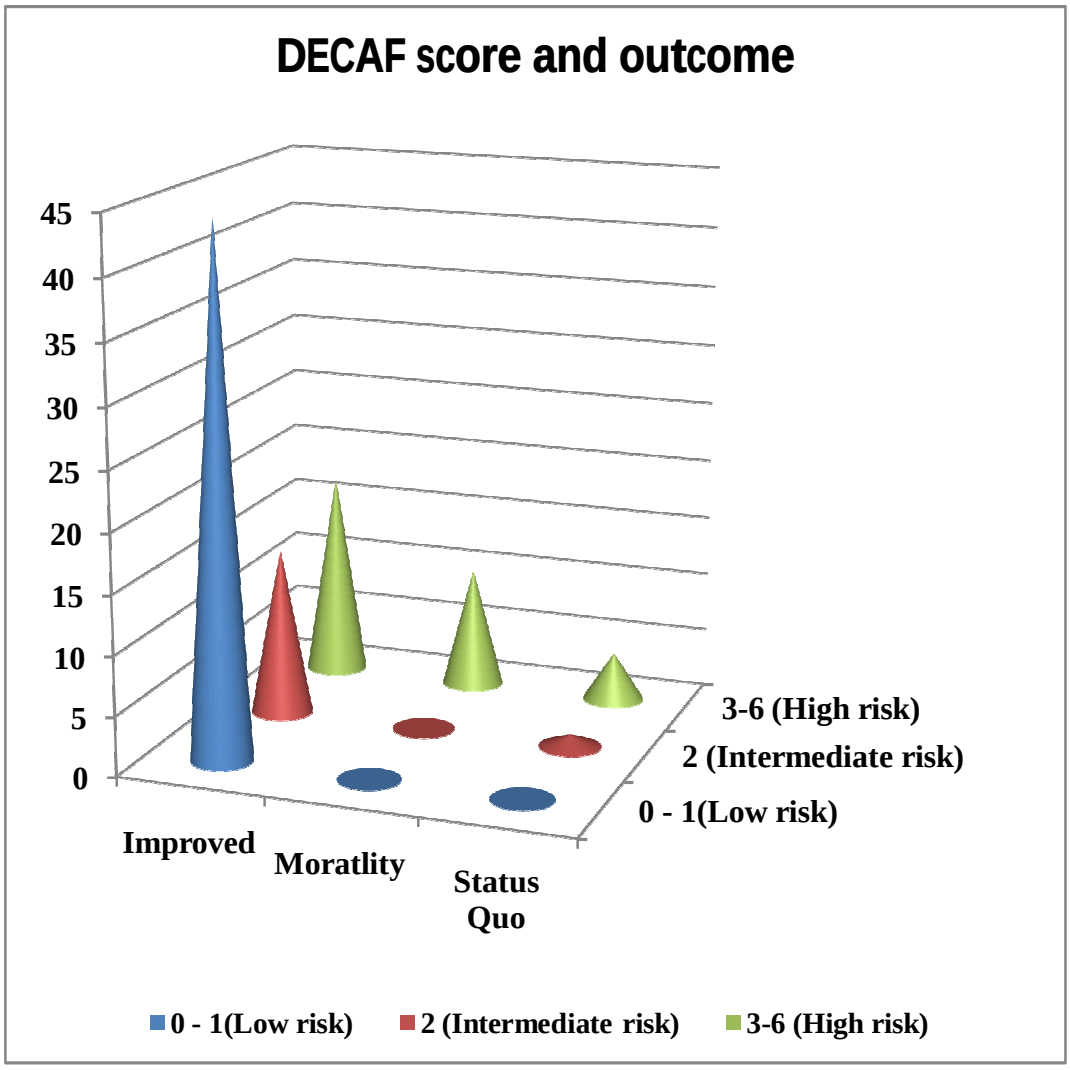
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	**6.488[a]**	**2**	**.039**
Likelihood Ratio	4.276	2	.118
Linear-by-Linear Association	5.106	1	.024
N of Valid Cases	90		

a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is .22.

Significant association exists between atrial fibrillation and outcome.

The DECAF score and outcome:

The DECAF score comprising the five variables – Dyspnea, Eosinopenia, Consolidation, Acidemia, atrial Fibrillation is strongly associated with outcome. There is no mortality in the in patients with DECAF score between 0-2. The mortality rate for patients getting score of 3 and above is 10 out of 31. In terms of percentage this is 32.3%. The higher is the DECAF score , the higher is the mortality. This relation is statistically significant at p=0.000.



DECAF Score Vs Outcome	Improved	Moratlity	Status Quo	Total
0 - 1(Low risk)	44	0	0	44
2 (Intermediate risk)	14	0	1	15
3-6 (High risk)	17	10	4	31
Total	75	10	5	90

Fig 20 : DECAF score and outcome

Table 24 : Crosstab DECAF and outcome

			DECAF						Total
			0	1	2	3	4	5	
outcome	improved	Count	31	13	14	10	6	1	75
		Expected Count	25.8	10.8	12.5	11.7	12.5	1.7	75.0
		% within outcome_effect	41.3%	17.3%	18.7%	13.3%	8.0%	1.3%	100.0%
		% within DECAF	100.0%	100.0%	93.3%	71.4%	40.0%	50.0%	83.3%
	status_quo	Count	0	0	1	4	0	0	5
		Expected Count	1.7	.7	.8	.8	.8	.1	5.0
		% within outcome_effect	.0%	.0%	20.0%	80.0%	.0%	.0%	100.0%
		% within DECAF	.0%	.0%	6.7%	28.6%	.0%	.0%	5.6%
	mortality	Count	0	0	0	0	9	1	10
		Expected Count	3.4	1.4	1.7	1.6	1.7	.2	10.0
		% within outcome_effect	.0%	.0%	.0%	.0%	90.0%	10.0%	100.0%
		% within DECAF	.0%	.0%	.0%	.0%	60.0%	50.0%	11.1%
Total		Count	31	13	15	14	15	2	90
		Expected Count	31.0	13.0	15.0	14.0	15.0	2.0	90.0
		% within outcome_effect	34.4%	14.4%	16.7%	15.6%	16.7%	2.2%	100.0%
		% within DECAF	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

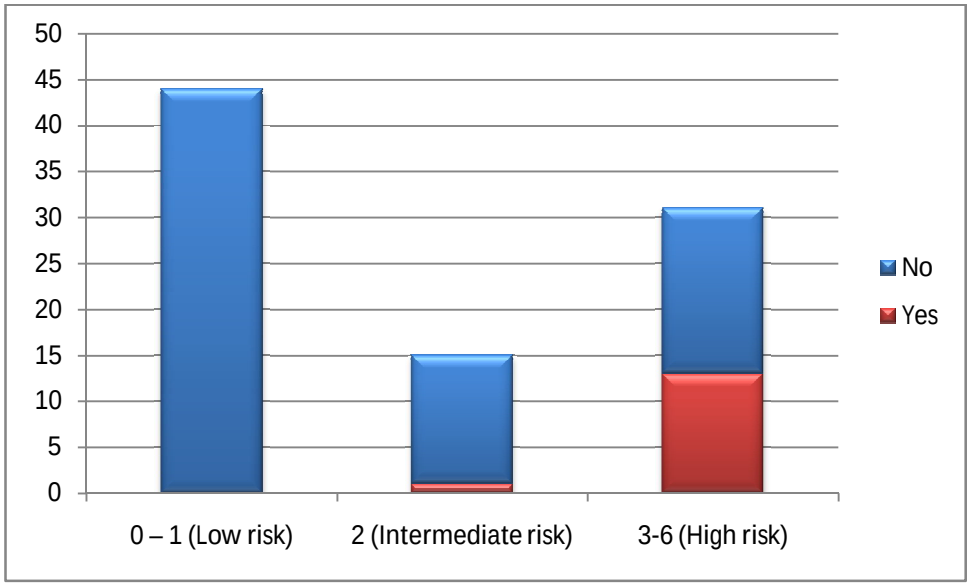
	Value	DF	Asymp. Sig. (2-sided)
Pearson Chi-Square	65.403[a]	10	.000
Likelihood Ratio	53.134	10	.000
Linear-by-Linear Association	29.522	1	.000
N of Valid Cases	90		

a. 13 cells (72.2%) have expected count less than 5. The minimum expected count is .11.

Significant association exists between DECAF score and outcome

DECAF score and use of ventilator:

As the DECAF score increases the need for ventilator use increases. In the low and intermediate risk group (DECAF 0-2) only one patient was ventilated. In the high risk group 13 out of 31 persons were ventilated. In terms of percentage this is 1.7% and 41% in the low-intermediate risk group and high risk group respectively. This association is statistically significant at p=0.000.



DECAF Score Vs Use of Ventilator	Yes	No	Total	P-value
0 – 1 (Low risk)	0	44	44	**0.000(S)**
2 (Intermediate risk)	1	14	15	
3-6 (High risk)	13	18	31	
Total	14	76	90	

Fig 21 : DECAF score and use of ventilator

By chi-square test, there is a significant association between the DECAF score and outcome,P≤0.05,(P=0.000).

DECAF score and duration of hospital stay:

The average duration of hospital stay for the low to intermediate risk group (DECAF 0-2) was 6.1. Whereas the average duration of hospital stay for the high risk group (DECAF 3-6) is 9.3. The higher is the DECAF score, the longer is the hospital stay. This association between the DECAF score and in-hospital stay is statistically significant at p=0.01.

Table 25 : Cross tabulation DECAF Score and Hospital stay

Count		DECAF						Total
		0	1	2	3	4	5	
Association between DECAF Score and Hospital stay	upto 3 days	6	1	0	0	2	0	9
	upto 6 days	23	8	5	4	3	0	43
	upto 9 days	1	2	3	4	2	0	12
	upto12 days	0	1	3	4	3	1	12
	upto 15 days	1	1	3	2	3	1	11
	upto 18 days	0	0	1	0	2	0	3
Total		31	13	15	14	15	2	90

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	**44.865[a]**	**25**	**.009**
Likelihood Ratio	51.370	25	.001
Linear-by-Linear Association	23.808	1	.000
N of Valid Cases	90		

a. 31 cells (86.1%) have expected count less than 5. The minimum expected count is .07.

Chi-square value 44.87 for DF 25 was found to be statistically significant at 0.01 and 0.05 level. From the results it was observed that significant association exists between the DECAF score and Hospital stay.

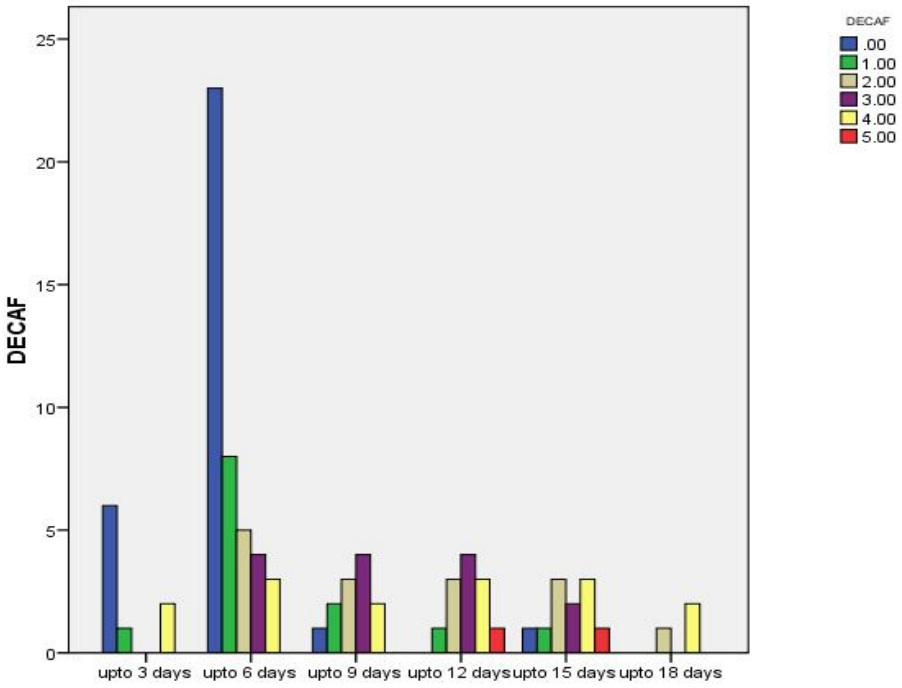


Fig 22: DECAF score and hospital stay

AGE and DECAF score:

The number of patients less than 60 years of age in the low, intermediate and high risk groups are 18, 12 and 18 respectively. The number of patients more than 60 years of age in the low, intermediate and high risk groups are 26, 3 and 13 respectively. The age distribution of low, intermediate and high risk groups is shown in the table. There is no significant relation between age and DECAF score.

Table 26 : Age and DECAF score

Age	DECAF score			Total	P-value
	Low risk (0-1)	Intermediate risk (2)	High risk (3-6)		
<=40	0	2	1	3	
40-50	8	4	8	20	0.209(NS)
50-60	10	6	9	25	
60-70	15	3	8	26	
70-80	10	0	5	15	
>80	1	0	0	1	
Total	44	15	31	90	

Gender and DECAF score:

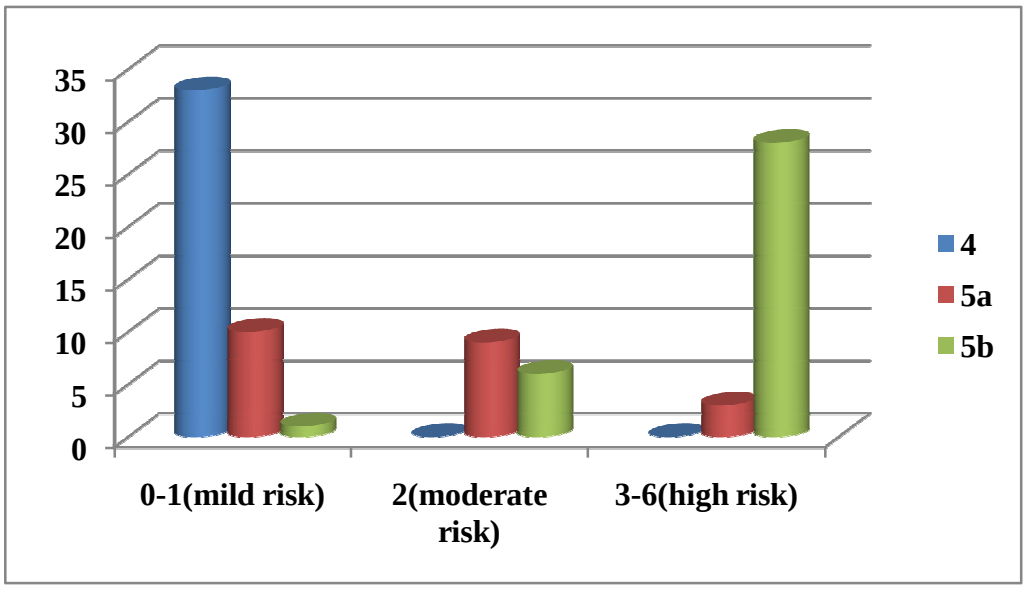
The number of female patients in low, intermediate and high risk groups are 4,2 and 3 respectively. In terms of percentage this is 44, 22.2 and 33.3 respectively. The number of male patients in low, intermediate and high risk groups are 40, 13 and 28 respectively. In terms of percentage this is 49.4, 16 and 34.6 respectively. There was no significant association between DECAF score and gender.

Table 27 : Gender and DECAF score

Gender	DECAF score			Total	P-value
	Low risk (0-1)	Intermediate risk (2)	High risk (3-6)		
Female	4	2	3	9	
Male	40	13	28	81	0.892(NS)
Total	44	15	31	90	

Dyspnea grade and DECAF score:

In patients with dyspnea grade 4 all 33 patients were in low risk group. In patients with dyspnea grade 5a the number of patients in low, intermediate and high risk groups are 10,9 and 3 respectively. In terms of percentage this is 45.5, 41 and 13.6 respectively. In patients with dyspnea grade 5b the number of patients in low, intermediate and high risk groups are 1,6 and 28 respectively. In terms of percentage this is 2.9, 17.1 and 80 respectively. Patients with higher grade of dyspnea according to eMRC had higher DECAF score. Patients having a dyspnea grade of 4 and 5a had better prognosis than patients having a grade 5b. This association is statistically significant p=0.000.



Dyspnea Score Vs DECAF Score	DECAF Score			Total	P-value
	0-1	2	3-6		
4	33	0	0	33	**0.000(S)**
5a	10	9	3	22	
5b	1	6	28	35	
Total	44	15	31	90	

Fig 23 : Dyspnea score and DECAF score

By chi-square test, there is a significant association between the Dysnea Score and Decaf score, P≤0.05, (P= 0.000).

Corpulmonale and DECAF score:

In patients with corpulmonale the number of patients in low, intermediate and high risk groups are 4,2 and 16 respectively. In terms of percentage this is 18.2, 9.1 and 72.7 respectively. In patients without corpulmonale the number of patients in low, intermediate and high risk groups are 40, 13 and 15 respectively. In terms of percentage this is 58.8, 19.1 and 22.1 respectively. Patients with corpulmonale had higher DECAF score. Most of the patients without pulmonary hypertension had a score of 0 to 2 with good prognosis. This association between DECAF score and corpulmonale is statistically significant at p=0.03.

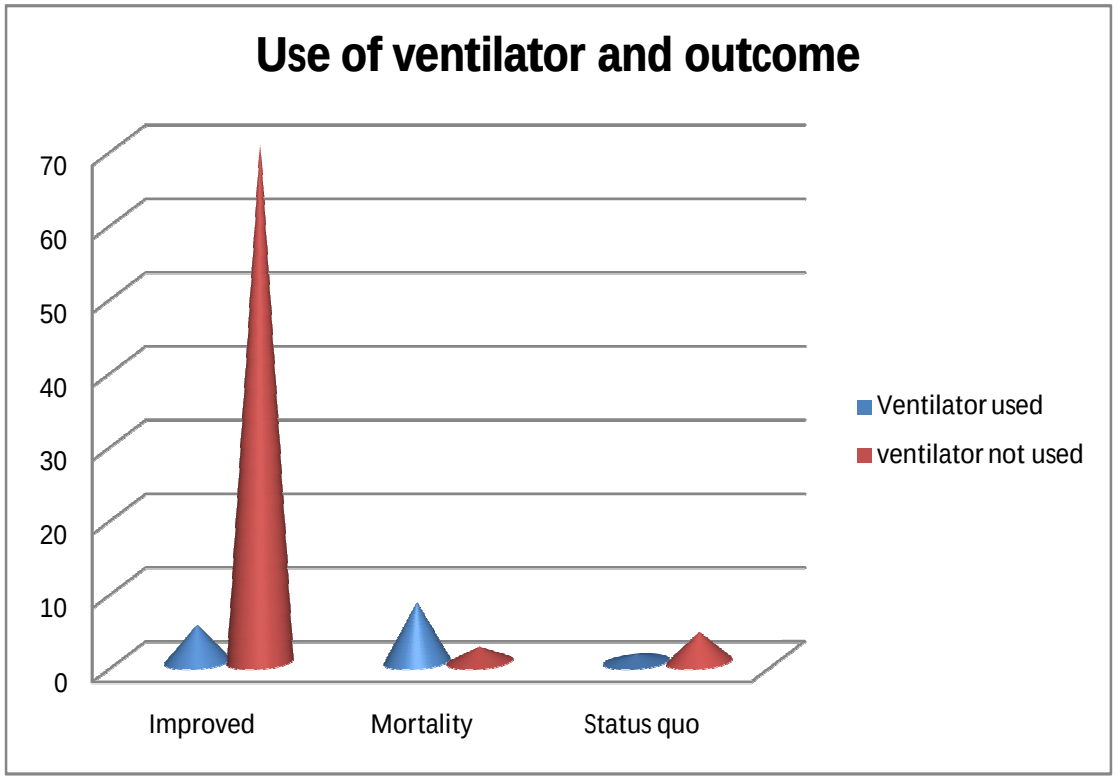
Table 28 : Corpulmonale and DECAF score

Cor pulmonale	**DECAF score**			**Total**	**P-value**
	Low risk (0-1)	Intermediate risk (2)	High risk (3-6)		
Mild PHT	3	2	4	9	
Moderate PHT	0	0	2	2	0.000(S)
Severe PHT	1	0	10	11	
No PHT	40	13	15	68	
Total	44	15	31	90	

By chi-square tests, there is a significant association between the Corpulmonale and DECAF score.

Use of ventilator and mortality:

The mortality rate among patients who were ventilated and those not ventilated are 57.1% and 2.6% respectively. There was higher mortality among patients who were ventilated. This relation between use of ventilator and outcome is statistically significant at p=0.000.



Use of ventilator	Outcome			Total
	Improved	Mortality	Status quo	
Yes	5	8	1	14
No	70	2	4	76
Total	75	10	5	90

Fig 24 : Use of ventilator and outcome

Table 29 : Crosstab – use of ventilator and outcome

			Ventilator		Total
			yes	No	
outcome	Improved	Count	5	70	75
		Expected Count	11.7	63.3	75.0
		% within outcome_effect	6.7%	93.3%	100.0%
		% within ventilation	35.7%	92.1%	83.3%
	status quo	Count	1	4	5
		Expected Count	.8	4.2	5.0
		% within outcome_effect	20.0%	80.0%	100.0%
		% within ventilation	7.1%	5.3%	5.6%
	Mortality	Count	8	2	10
		Expected Count	1.6	8.4	10.0
		% within outcome_effect	80.0%	20.0%	100.0%
		% within ventilation	57.1%	2.6%	11.1%
Total		Count	14	76	90
		Expected Count	14.0	76.0	90.0
		% within outcome_effect	15.6%	84.4%	100.0%
		% within ventilation	100.0%	100.0%	100.0%

CHI-SQUARE TESTS

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	36.203[a]	2	.000
Likelihood Ratio	26.049	2	.000
Linear-by-Linear Association	34.006	1	.000
N of Valid Cases	90		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .78

Significant association is exist between outcome and use of ventilation

Hospital stay and outcome:

The average hospital stay for study population was 7. Longer in-hospital stay was not associated with better prognosis. There was no significant relationship between duration of hospital stay and outcome.

Table 30 : Hospital stay and outcome

Hospital stay	Outcome			Total	P-value
	Improved	Mortality	Status quo		
>5 days	30	1	1	32	
5-10 days	27	3	2	32	0.270(NS)
10-15 days	16	5	2	23	
>=15 days	2	1	0	3	
Total	75	10	5	90	

By chi-square test, there is no significant association between hospital stay and outcome, P≥0.05,P=0.270.

Ventilator use and hospital stay :

The average duration of hospital stay for patients who were ventilated and not ventilated is 11.1 and 6.5 respectively. Patients who were put on ventilator had longer hospital stay, compared to patients not ventilated. This association is statistically significant at p=0.001

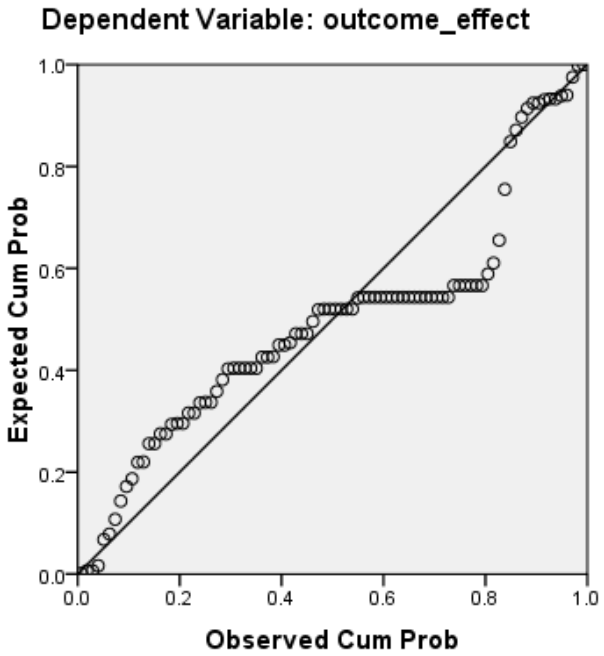
Table 31 : Use of ventilator and hospital stay

Hospital Stay		Use of Ventilator		Total	
		Yes	No		P-value
	<5days	1	31	32.0	
	5to 10 days	3	29	32.0	**0.001(S)**
	10 to 15 days	8	15	23.0	
	>15 days	2	1	3.0	
Total		14.0	76.0	90.0	

By chi-square test, there is a significant association between hospital stay and use of ventilator, P≤0.05, P=0.001.

Prediction analysis on outcome based on the DECAF score and use of ventilator.

Fig 25 : Normal P-P Plot of Regression Standardized Residual



In predicting the outcome, the influence of DECAF and use of ventilation is a significant one . The R-Square value of 0.477 explains 47 percent of total variance. It has been confirmed by the f-value of 26.17 which was significant at 0.05 level and confirms that not occurred by a chance.

DISCUSSION

Clinical Profile of the study population:

A total of 90 patients were included in our study as per our patient selection methods, inclusion and exclusion criteria. The age group of our patients in our study ranged from 37 to 82. The number of patients in the age groups ≤40, 40-50, 50-60, 60-70, 70-80, >80 were 3 (3.3%), 20 (22.2%), 25 (27.8 %), 26 (28.9%), 15(16.7%) and 1(1.1%) respectively. This distribution shows that we had more patients in older age groups than younger age groups. This is consistent with the fact that age is often listed as a risk factor for COPD[49]. It is unclear if healthy aging as such leads to COPD or if age reflects the sum of cumulative exposures throughout life. Beyond 70 years of age there are fewer study subjects. This situation may have arisen because of exclusion of patients with other co-morbidities. Since co-morbid illnesses are common with aged population we had this sort of age distribution of patients.

Out of the 90 patients in the study, 81 are male and 9 are female. Thus males accounted for 90% of our study population while females accounted for 10%. This could be attributed to low prevalence of smoking among ladies. This shows that smoking habit may not have entered into our female population as much as in the western literature. Another reason could be that many female patients with COPD are usually branded as having asthma in our country. The primary reason for

females developing COPD in our country could be attributed to passive smoking, biomass exposure and post tuberculosis.

In the study population, 47 did not have any comorbid illness. The most common comorbidity among the study population was Pulmonary Tuberculosis Sequalae. 22 patients had history and radiological features of prior pulmonary tuberculosis. 6 patients had systemic hypertension, 2 patients had chronic kidney disease, 1 patient had both hypertension and diabetes mellitus, 4 patients had coronary artery disease, 2 patients had obstructive sleep apnea, 3 patients had connective tissue disorders, 3 patients had hypersensitivity pneumonitis. This is consistent with the finding that tuberculosis has been found to be risk factor for COPD[1]. In addition tuberculosis is a potential comorbidity in COPD patients. Severe respiratory infections have been associated with reduced lung function. Three patients in the study had coexistent hypersensitivity pneumonitis. Estimates by American Thoracic Society concluded that occupational exposures account for 10-20% of either symptoms or functional impairment consistent with COPD[50]. The risk from occupational exposures in less regulated areas like India is likely to be much higher than reported in Western literature.

Corpulmonale is classically defined as “hypertrophy of the right ventricle resulting from diseases affecting the function and/or structure of the lungs except when these pulmonary alterations are the result of

diseases that primarily affect the left side of the heart"[51]. Out of 90 patients included in the study 22 (24.4%) patients had corpulmonale as evidenced on echo. Out of them 9 (10%) patients had mild pulmonary hypertension (PHT), 2 (2.2%) patients had moderate PHT, 11 (12.2%) patients had severe PHT. This is consistent with other studies that have reported prevalence varying considerably from 20%–91%[52,53] depending on the definition of pulmonary hypertension, the severity of lung disease in the group studied and the method of measuring the pulmonary artery pressure (PAP). During an exacerbation of COPD, PAP may rise by as much as 20 mm Hg and return to its baseline after recovery[53]. Pulmonary hypertension in COPD has been considered to be the result of hypoxic pulmonary vasoconstriction, polycythemia and destruction of the pulmonary vascular bed by emphysema. Recently, it has been recognized that hyperinflation and endothelial dysfunction also play a role in the pathogenesis of PH.

MRC dyspnea scale is used for dyspnea grading because it is simple and allows patients to indicate the level of breathlessness. Extended Medical Research Council (eMRC) is used since it includes functional dependence as well. When dyspnea was graded according to eMRC grades, 33 patients had eMRC grade 4, 22 patients had eMRC grade 5a and 35 patients had a score of 5b. In terms of percentage, the distribution of patients in grades 4, 5a and 5b was 36.7, 24.4 and 38.9

respectively. Since patients were admitted with acute exacerbation of COPD most of them had dyspnea at rest, grade 5 which was again subdivided into 5a and 5b based on functional dependence. The patients with grade 4 dyspnea, though they did not have dyspnea at rest, they were admitted for AECOPD with increased quantity and purulency of sputum.

Eosinopenia was defined as an absolute eosinophil count of less than 50/mm^3. 6 out of 90 patients had eosinopenia. Hence 6.7% of the study population had low eosinophil count. The incidence of eosinopenia is comparatively lower in our study population compared to western literature. It has been shown in previous studies that eosinopenia accompanies the response to acute infection and inflammation[22]. Thus in AECOPD eosinopenia may reflect severity of accompanying acute inflammatory response.

Assessment of chest radiographs of patients at admission to confirm the presence of consolidation was done. Accordingly 25 (27.77%) patients had consolidation on chest radiograph. Similar prevalence of 32.5% of consolidation in patients with AECOPD was reported by J Steer et al[19] in his study of 920 patients. In two UK national audits[26,27], consolidation was reported in 16% of all admissions and in 34% of patients requiring ventilator assistance. Many a times the

cause for an acute exacerbation of COPD is infectious and related to viral or bacterial infection.

Out of 90 patients 25 patients (27.77%) had acidemia. This is consistent with various studies that have reported a prevalence between 25% to 53%[19,21]. In a study involving consecutive patients admitted with AECOPD over one and half year period in UK, the incidence of acidemia was 27.9%.

Respiratory acidosis in COPD is secondary to hypoventilation. It includes multiple mechanisms including decreased responsiveness to hypoxia and hypercapnia, increased ventilation-perfusion mismatch leading to increased dead space ventilation and decreased diaphragmatic function due to fatigue and hyperinflation.

Among the study population 4 (4.4%) patients had atrial fibrillation, while the remaining 86 did not have fibrillation. This incidence is lower compared to other studies which have reported an occurrence of above 12%[39]. Acidemia, drugs and corpulmonale contribute to occurrence of arrythmias in COPD patients. It is postulated that ectopic beats initiating atrial fibrillation originate in the walls of pulmonary veins and it could be triggered by changes in gas composition[38].

The average duration of hospital stay for the study group was 7 days. The length of hospital stay was divided into four groups- <5 days,

5-10 days, 10-15 days, >15 days. 32 patients had hospital stay lesser than 5 days, 32 patients had hospital stay between 5-10 days, 23 patients had hospital stay between 10-15 days, 3 patients stayed in hospital longer than 15 days. In the study by Ying et al[54] in Oslo involving 590 patients admitted with AECOPD, the median length of hospital was 6 days. Various studies have illustrated wide range of hospital stay between 3-11 days.

The mortality rate for the study population was 10 out of 90 (11%). 75 patients "improved" at the time of discharge, 10 patients died in the hospital, 5 patients were discharged against medical advice whose clinical condition could not be defined as 'improved' or deteriorated at the time of leaving the hospital. This study confirms the findings of previous studies. Karin H Groenewegen et al[33], in a study of 171 patients admitted with AECOPD showed the mortality rate during hospital stay was 8%, increasing to 23% after 1 year of follow-up. In the study by J Steer et al[19] the in-hospital mortality rate was 10.4% (96/920) . In the study by Connors et al[55] the in-hospital mortality was 11%.

Out of the 90 patients 14(15.6%) were put on ventilator, 6 were put on non invasive ventilation and 8 were put on Invasive ventilation. This is consistent with the previous studies that have reported ventilator use between 8 to 12% in patients getting admitted with AECOPD[32,33].

Factors influencing in-hospital prognosis:

The mortality in the age groups 40-50, 50-60, 60-70, 70-80 is 4,4,1,1 respectively. When age was taken as scattered variables, older patients showed higher mortality. However there is no statistically significant association between age and outcome,P≥0.05(P=0.800). The number of patients put on ventilator in the age groups 40-50, 50-60, 60-70 is 7, 6, 1 respectively. In the study group, the use of ventilator increases as the age advances. None of the patients less than 40 years or above 70 years were put on ventilator. The use of ventilator is more with older patients. This relationship is statistically significant at p=0.020. In the study population the average number of hospital stay is 7 days. It is seen that as age advances the duration of hospital stay increases with older people having the need to stay longer. This association is statistically significant at p=0.055 (by ANOVA). These findings show that younger patients have milder forms of the disease compared to older patients. This could be attributed to decline in lung function as age advances and presence of comorbid illness which are more in older patients.

The mortality among female patients in the study is 2 out of 9 (18%). The mortality among the male patients is 8 out of 68 (11.8%).There is no significant association between gender and outcome. This could be attributed to the very low number of female participants in the study. Male patients had longer in-hospital stay compared to female

patients. This association is statistically significant at p=0.009. 12 out of 69 (17.4%) male patients were put on ventilator whereas 2 out of 9 (22.2%) female patients were ventilated. There is no statistically significant gender difference in the use of ventilator among the study population. This agrees with the findings by S Yu et al[56] and Cooper et al[57] who showed that gender was not an independent risk factor for short or long term prognosis in acute exacerbation of COPD.

The mortality among patients with corpulmonale is 27.3 % (6/22). The mortality among patients without corpulmonale is 5.9% (4/68). Among the study population, patients having corpulmonale and pulmonary hypertension had higher mortality. Most of the patients who did not have PHT improved. This association between corpulmonale and outcome is statistically significant at p=0.015. 9 out of 22 (41%) patients with corpulmonale required ventilator support. 5 out of 63 (8%) persons without corpulmonale required ventilator support. There was increased need for usage of ventilators in patients with pulmonary hypertension compared to patients without PHT. This relationship was statistically significant at $p<0.05$. Most of the patients without PHT didn't require ventilator. Patients with corpulmonale had a longer duration of hospital stay. Patients without corpulmonale stayed for only 5-10 days. This relation was statistically significant at p=0.03. This finding agrees with

various studies by JJ Soler et al[58], M Oswald et al[59] which have showed that corpulmonale is an adverse prognostic variable in AECOPD.

In patients getting admitted with AECOPD, as the eMRC dyspnea grade increases, the mortality increases. Almost all the patients in the score of 4 and 5a got improved. Mortality is predominantly seen in eMRC 5b group. The mortality rate among eMRC 5b is 10 out of 35(28.6%). The relation is statistically significant at p=0.000. With increasing grade of dyspnea there is increased usage of ventilator support. None of the patients in the score of 4 and 5a required ventilator whereas 66% in the dyspnea score group of 5b required ventilator. This association is statistically significant at p=0.000. With increasing grade of dyspnea, the in-hospital stay for patients admitted with AECOPD increases . This association is statistically significant at p=0.000. In the study by E Steer et al[21] the in hospital mortality rate for eMRCD 5b patients was 33.1%. The findings of the present study are consistent with previous studies[1921] which have showed that severity of dyspnea is strongly associated with both in-hospital mortality and early readmission. By combining MRCD scale with a person's ability to manage personal care (eMRCD) the predictive value of dyspnea scoring is improved.

All the 6 patients with eosinopenia improved. None of the patients with eosinopenia died during hospital stay. In our study there is no

significant association between eosinopenia and outcome. The finding of our study is not comparable with other studies by Holland et al[25] and J steer et al[19] that have showed eosinopenia to be a significant prognostic factor in AECOPD.

9 out of the 10 (90%) patients in the mortality group had chest X-ray features of consolidation. Out of 75 patients who improved 16 (21.3%) had consolidation. Presence of consolidation is associated with higher mortality. This association is statistically significant at p=0.000. This is comparable to previous studies by Liebermann et al, J Steer et al who have shown that mortality among pneumonia associated AECOPD is more than non pneumonic AECOPD. Community acquired pneumonia is common among patients hospitalised with AECOPD and usually causes the exacerbation to have more severe clinical and laboratory parameters.

All 10 (100%) patients with in-hospital mortality had arterial blood pH<7.30. 14 out of 75(18.7%) patients who improved had acidemia. Presence of acidemia is associated with higher in-hospital mortality. This relation is statistically significant at p=0.00. According to previous studies[56], the frequency of hypercapnic respiratory failure in patients with AECOPD varies from 16-35% with overall mortality of 35-43%[5]. Our study has shown higher mortality among hypercapnic patients compared to previous studies. The level of hypercapnia, suggestive of chronic alveolar hypoventilation, reflects the severity of the

underlying respiratory condition, and Patients with chronic hypercapnia, who comprised the majority of our study population, have a worse prognosis than patients with normoventilation.

In the study group 4 patients had atrial fibrillation. All of them died during the course of treatment. In terms of percentage, 20% of the patients with in-hospital mortality had atrial fibrillation. Presence of atrial fibrillation is associated with higher mortality. By Chi square this relation is statistically significant at p=0.03. This is in accordance with study by J Steer et al[19] in which 26% of patients with in hospital mortality had atrial fibrillation. Previous studies have shown that occurrence of atrial fibrillation is associated with poor prognosis.

The average hospital stay for study population was 7. Longer in-hospital stay was not associated with better prognosis. There was no significant relationship between duration of hospital stay and outcome. There are no clinical trials that have evaluated the optimal duration of treatment in AECOPD. Our study shows that prolonged hospital stay is not necessarily associated with improved outcome.

The mortality rate among patients who were ventilated and those not ventilated are 57.1% and 2.6% respectively. There was higher mortality among patients who were ventilated. This relation between use of ventilator and outcome is statistically significant at p=0.000. The average duration of hospital stay for patients who were ventilated and not

ventilated is 11.1 and 6.5 respectively. Patients who were put on ventilator had longer hospital stay, compared to patients not ventilated. This association is statistically significant at p=0.001. Out of 14 patients who required ventilatory support, 8 were put on invasive ventilation and 6 were put on non invasive ventilation. Use of ventilator improves acute respiratory acidosis, decreases respiratory rate, work of breathing, severity of breathlessness. However the higher incidence of mortality and longer hospital stay in patients put on ventilator may be attributed to the fact that weaning or discontinuation from mechanical ventilation can be difficult and hazardous in patients with COPD[1]. In patients on ventilatory support there is higher incidence of ventilator associated pneumonia, barotraumas and need for longer antibiotic usage. This study highlights the need for trial of non invasive ventilation even in conditions where invasive ventilation is generally indicated. NIV can reduce the complications associated with intubation like ventilator associated pneumonia.

The use of DECAF Score in assessing in-hospital prognosis:

Out of 90 patients studied, 44 patients had a DECAF score between 0-1 (low risk), 15 patients had a DECAF score of 2 (intermediate risk) and 31 patients had a DECAF score between 3-6(high risk). In terms of percentage this is 48.9%, 16.7% and 34.4% respectively. This is consistent with the study by J.Steer et al[19] , in which the low risk group

comprised 53.5% of the study population, intermediate risk group comprised 24.5%of the study population, high risk group comprised 22% of the study population. This shows that in a given population getting admitted with AECOPD, low risk group outnumber the high risk patients. This may be due to the fact that these patients approach health care facilities early during the course of exacerbation. The DECAF score comprising the five variables – Dyspnea, Eosinopenia, Consolidation, Acidemia, atrial Fibrillation is strongly associated with outcome. There is no mortality in the in patients with DECAF score between 0-2. The mortality rate for patients getting score of 3 and above is 10 out of 31. In terms of percentage this is 32.3%. The higher is the DECAF score , the higher is the mortality. This relation is statistically significant at p=0.000. Our study agrees with the findings by J Steer et al[19]. In their study involving 920 AECOPD patients, the strongest five categorical variables strongly associated with in-hospital mortality were selected and the DECAF score devised. They reported that in DECAF 0-1 the in-hospital mortality was 1.4%, in DECAF 2 mortality was 8.4%. and in DECAF 3-6 the mortality was 34.6%. As the DECAF score increases use of ventilator increases. In the low and intermediate risk group (DECAF 0-2) 1 out of 59 persons was ventilated. In the high risk group 13 out of 31 persons were ventilated. In terms of percentage this is 1.7% and 41% in the low-intermediate risk group and high risk group respectively. This

association is statistically significant at p=0.000. The average duration of hospital stay for the low to intermediate risk group (DECAF 0-2) was 6.1. whereas the average duration of hospital stay for the high risk group (DECAF 3-6) is 9.3. The higher is the DECAF score the longer is the hospital stay. This association between the DECAF score and in-hospital stay is statistically significant at p=0.01. To our knowledge there are no previous studies that have evaluated the association between DECAF score and need for ventilator use or duration of hospital stay.

Factors related to DECAF score:

The number of patients less than 60 years of age in the low, intermediate and high risk groups are 18, 12 and 18 respectively. The number of patients more than 60 years of age in the low, intermediate and high risk groups are 26, 3 and 13 respectively. There is no significant relation between age and DECAF score. The number of female patients in low, intermediate and high risk groups are 4,2 and 3 respectively. In terms of percentage this is 44, 22.2 and 33.3 respectively. The number of male patients in low, intermediate and high risk groups are 40, 13 and 28 respectively. In terms of percentage this is 49.4, 16 and 34.6 respectively. There was no significant association between DECAF score and gender.

In patients with dyspnea grade 4 all 33 patients were in low risk group. In patients with dyspnea grade 5a the number of patients in low,

intermediate and high risk groups are 10,9 and 3 respectively. In patients with dyspnea grade 5b the number of patients in low, intermediate and high risk groups are 1,6 and 28 respectively. Patients having a dyspnea grade of 4 and 5a had better prognosis than patients having a grade 5b. Patients with higher grade of dyspnea according to eMRC had higher DECAF score. This association is statistically significant p=0.000. In patients with corpulmonale the number of patients in low, intermediate and high risk groups are 4,2 and 16 respectively. In terms of percentage this is 18.2, 9.1 and 72.7 respectively. In patients without corpulmonale, the number of patients in low, intermediate and high risk groups are 40, 13 and 15 respectively. In terms of percentage this is 58.8, 19.1 and 22.1 respectively. Patients with corpulmonale had higher DECAF score. Most of the patients without pulmonary hypertension had a score of 0 to 2 with good prognosis. This association between DECAF score and corpulmonale is statistically significant at p=0.03. Hence higher dyspnea grade and presence of corpulmonale may be taken as indirect markers of higher DECAF score.

CONCLUSION

- The present study shows that in patients admitted with acute exacerbation of COPD the DECAF score comprising the five variables – Dyspnea, Eosinopenia, Consolidation, Acidemia, atrial Fibrillation is strongly associated with outcome.
- Based on DECAF score these patients are divided into low risk (DECAF 0-1), intermediate risk (DECAF-2) and high risk (DECAF 3-6).
- The higher the DECAF score , the higher is the mortality, the longer is the hospital stay and the higher is the need for use of ventilator.
- Presence of corpulmonale can be considered as a surrogate marker of higher DECAF score.

The DECAF score is a simple clinical tool for assessing in-hospital prognosis in patients admitted with acute exacerbation of Chronic Obstructive Pulmonary Disease. This scoring system incorporates indices routinely available and can stratify patients admitted with AECOPD into clinically relevant risk groups.

Hence assessing the DECAF score at the time of admission in AECOPD helps in decision regarding

1. Early escalation of care
2. Deciding the location of treatment – Intensive care or ward
3. Determining the need for use of ventilator
4. Deciding on end-of-life care
5. Helps the physician in informing the patient and relatives regarding the prognosis and exacerbation related short term risks.

LIMITATIONS

The major limitations of our study are:

1). Lack of post hospital follow up data, which would be necessary for validation of predictive factors found in the present study.

2). The number of female patients enrolled in the study was quite small, lesser than that expected. However since consecutive patients were recruited, this has to be considered as corresponding to what occurs in the real life setting.

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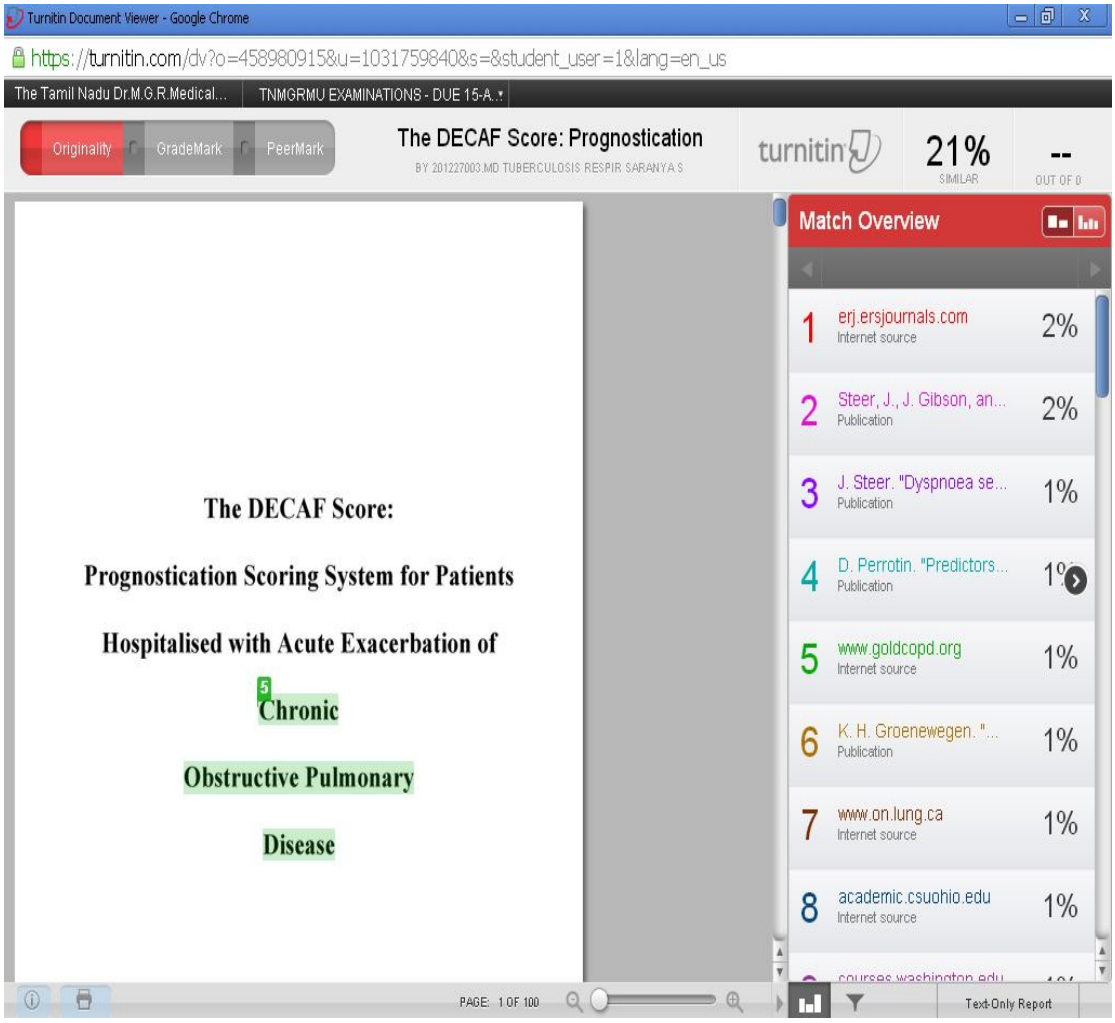
ABBREVIATIONS

AECOPD	Acute Exacerbation of Chronic Obstructive Pulmonary Disease
AF	Atrial Fibrillation
COPD	Chronic Obstructive Pulmonary Disease
DECAF	Dyspnea, Eosinopenia, Consolidation, Acidemia, Fibrillation.
eMRCD	extended Medical Research Council Dyspnea Score
FEV1	Forced Expiratory Volume in one second
FVC	Forced Vital Capacity
MRCD	Medical Research Council Dyspnea Score
NPAE	Non pneumonic Acute Exacerbation
PNAE	Pneumonic Acute Exacerbation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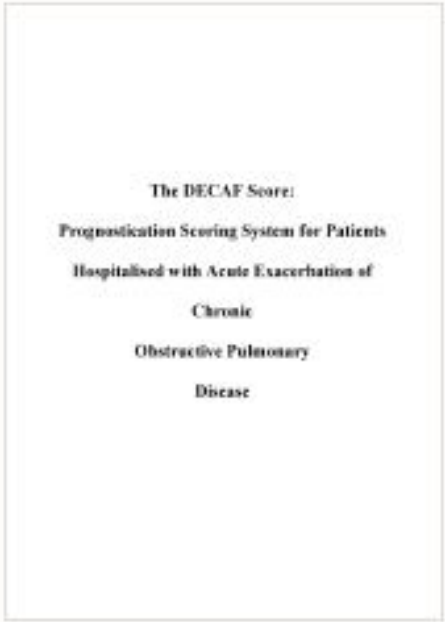

The DECAF Score:

Prognostication Scoring System for Patients

Hospitalised with Acute Exacerbation of

Chronic

Obstructive Pulmonary

Disease

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI – 3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No: 044 25305301
Fax: 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.Saranya.S
PG in Thoracic Medicine,
Institute of Thoracic Medicine,
Madras Medical College, Chennai – 3.

Dear Dr Saranya.S,

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled, **"The DECAF Score – Prognostication Scoring System for Patients Hospitalized with Acute Exacerbation of Chronic Obstructive Pulmonary Disease."** No. 32032014

The following members of Ethics Committee were present in the meeting held on 11.03.2014 conducted at Madras Medical College, Chennai – 3.

1. Dr.C.Rajendran,M.D.	-	Chairperson
2. Prof.Kalaiselvi, M.D. Vice-Principal, MMC,Ch-3.	-	Member Secretariat
3. Prof. Nandhini, M.D. Institute of Pharmacology, MMC, Ch-3.	-	Member
4. Prof.Bhavani Shankar,M.S. Prof and HOD of General Surgery, MMC,CH-3.	-	Member
5. Prof. V. Padmavathi, M.D. I/c Director of Pathology, MMC, Ch-3.	-	Member
6. Thiru.S.govindasamy, BABL	-	Lawyer
7. Tmt.Arnold Saulina, MA MSW	-	Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

9/4/14

Member Secretary – Ethics Committee

VICE PRINCIPAL
MADRAS MEDICAL COLLEGE
CHENNAI-3.

PATIENT CONSENT FORM & INFORMATION SHEET

Title of the Project

The DECAF Score: Prognostication Scoring System for Patients Hospitalised with Acute Exacerbation of Chronic Obstructive Pulmonary Disease"

Institution : **Department of Thoracic Medicine,**
Madras Medical College,
Chennai-600 003.

Name	:	Date	:
Age	:	IP No	:
Sex	:	Project Patient No	:

The details of the study have been provided to me in writing and explained to me in my own language and the purpose of the study has been explained.

I confirm that I have understood the above study and had the opportunity to ask questions.

I understood that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected.

I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

I have been given an information sheet giving details of the study.

I fully consent to participate in the above study regarding '**Prognostication Scoring System for Patients Hospitalised with Acute Exacerbation of Chronic Obstructive Pulmonary Disease'**.

_____	_____	_____
Name of the Subject	Signature	Date
_____	_____	_____
Name of the Investigator	Signature	Date

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சி தலைப்பு : நாட்பட்ட நுரையீரல் காற்றுக்குழாய் அடைப்பு நோயினால் மருத்துவமனையில் அனுமதிக்கப்படும் நோயாளிகளுக்கு **DECAF Score** கொண்டு நோயின் விளைவுகளை முன்கணிப்பு செய்தல்.

பெயர் தேதி

வயது உள் நோயாளி எண்

பாலினம் ஆராய்ச்சி சேர்க்கை எண்

எனக்கு நாட்பட்ட நுரையீரல் காற்றுக்குழாய் அடைப்பு நோய் உள்ளதையும் அதன் விளைவுகளையும் விவரங்களையும் மருத்துவர் நன்கு தெரிவித்துள்ளார். மேற்கொள்ளப்படும் ஆராய்ச்சியின் விவரங்களையும் அதன் நோக்கங்களையும் முழுமையாக எனக்கு விளக்கப்பட்டுள்ளது. எனக்கு விளக்கப்பட்ட விவரங்களை நன்கு புரிந்துக்கொண்டு இந்த ஆராய்ச்சியில் பங்குக்கொள்வதற்கு சம்மதம் அளிக்கிறேன். இந்த ஆராய்ச்சியிற்காக இரத்த பரிசோதனை, சுருள்படும், மார்பகப்படம் எடுப்பதற்கும் சம்மதம் அளிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தம் இன்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துக்கொண்டேன். நான் என்னுடைய சுய நினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

கையொப்பம்.

Clinical Questionnaire

Name:

Age:

Sex:

IP/OP number:

Presenting illness:

Dyspnea Scoring:

Co morbidities:

Smoking history:

Clinical Examination:

Blood investigations:

Complete blood count:

Absolute Eosinophil Count:

Arterial Blood Gas analysis:

Radiological investigations:

DECAF Score :

Date of Discharge:

Duration of hospital stay:

Clinical Outcome: 1. Improved 2. Status Quo 3. Mortality

name	age	Sex	co morbidity	cor pulmonale	dyspnea	eosinopenia	consolidation	acidemia	fibrillation	DECAF score	hospital stay	outcome	use of ventilator
Thangadurai	64	male	nil	no	5b	no	yes	no	no	3	10	status quo	no
raman	56	male	nil	mild PHT	5a	no	yes	no	no	2	7	improved	no
susheela	75	female	nil	no	4	no	no	no	no	0	4	improved	no
selvan	55	male	DM,SHT	no	5b	no	no	no	no	2	13	improved	no
venkatesan	69	male	nil	no	5a	no	no	no	no	1	5	improved	no
vinayakam	60	male	CAD	no	5b	no	yes	yes	no	4	10	mortality	yes
abdul rashid	45	male	connective tissue disorder	mod PHT	5b	no	yes	yes	no	4	8	improved	no
subramani	65	male	SHT	severe pht	5b	no	yes	yes	no	4	2	improved	no
pooranam	67	female	PT SEQUALAE	mild PHT	4	no	no	no	no	0	4	improved	no
govindraj	70	male	SHT	no	5a	no	no	yes	no	2	13	improved	no
syed thajudeen	75	male	PT SEQUALAE	no	5b	no	yes	no	no	3	9	improved	no
damodaran	50	male	PT SEQUALAE	mild pht	5b	no	yes	yes	no	4	6	mortality	yes
singaram	72	male	PT SEQUALAE	no	5a	no	no	no	no	1	4	improved	no
selvaraj	71	male	SHT	no	5a	no	no	yes	no	3	10	improved	no
yakur sherif	70	male	SHT	mild PHT	5a	no	no	no	no	1	4	improved	no
Sengini	72	male	CKD	no	5a	no	no	no	no	1	6	improved	no
purushothaman	73	male	Nil	no	5b	no	no	yes	yes	4	5	mortality	no
ekambaram	60	male	nil	mild PHT	5a	no	yes	no	no	2	5	status quo	no
masilamani	75	male	SHT	no	5a	no	no	no	no	1	6	improved	no
perumal	60	male	PT SEQUALAE	mild PHT	5b	no	yes	yes	no	4	13	mortality	no
periasamy pillai	70	male	nil	no	5a	yes	no	yes	no	3	10	improved	no
meena	70	female	SHT	no	5a	no	no	no	no	1	10	improved	no
subramani	73	male	pt sequalae, ca stomach	mild PHT	4	no	no	no	no	0	14	improved	no
krishnan	62	male	nil	no	5a	yes	yes	no	no	3	5	improved	no
rose	50	female	PT SEQUALAE	no	5a	no	yes	no	no	2	5	improved	no
babu	60	male	PT SEQUALAE	no	5b	yes	yes	yes	no	5	11	improved	yes
balu	50	male	nil	no	5b	no	no	no	no	2	11	improved	no
sekar	47	male	osa	severe pht	5b	no	no	yes	no	3	12	status quo	yes
ananda sekaar	64	male	nil	no	5b	yes	no	no	no	3	4	improved	no
alagarsamy	63	male	nil	no	5a	no	no	yes	no	2	10	improved	no
nagarathinam	77	male	nil	no	5b	no	no	yes	yes	4	13	improved	no
navaneetham	50	female	SLE	mod PHT	5b	no	yes	yes	no	4	18	mortality	yes

vijayakumar	45	male	PT SEQUALAE	no	5b	no	no	no	no	2	6	improved	no

ganesan	75	male	CAD	no	4	no	no	no	no	0	6	improved	no
srinivasan	46	male	nil	no	5a	no	no	no	no	1	4	improved	no
murali	44	male	nil	no	5b	no	no	no	no	1	13	improved	no
aadhi	47	male	PT SEQUALAE	no	5a	no	no	yes	no	2	13	improved	no
sarangan	40	male	nil	no	5a	yes	no	no	no	2	7	improved	no
vajram	50	male	hypersenstivity pneumonitis	severe pht	5b	no	yes	yes	no	4	10	mortality	yes
murugan	30	male	PT SEQUALAE	no	5a	no	yes	no	no	2	11	improved	no
iyappan	47	male	PT SEQUALAE	no	4	no	no	no	no	0	9	improved	no
thiruneelakandan	67	male	nil	severe pht	5b	no	yes	yes	no	4	11	mortality	yes
mani	59	male	PT SEQUALAE	no	5b	no	yes	no	no	3	13	improved	no
kasiammal	47	female	nil	no	5b	no	yes	yes	no	4	9	mortality	yes
rajendran	60	male	ckd	no	5a	no	no	no	no	1	9	improved	no
kaliyan	65	male	nil	no	5a	no	no	no	no	1	4	improved	no
thanigavel	42	male	nil	severe pht	5b	no	no	yes	no	3	13	improved	yes
sundar raj	51	male	nil	severe pht	5b	no	yes	yes	yes	5	15	mortality	yes
karuna	58	male	nil	no	5b	no	no	no	yes	2	6	improved	no
ayyakannu	60	male	nil	mild PHT	5b	no	yes	no	no	3	8	status quo	no
sultan	63	male	OSA	no	5b	no	yes	no	no	3	4	status quo	no
chellan	55	male	PT SEQUALAE	severe pht	5b	no	yes	no	no	3	7	improved	yes
raguraman		male	pt seualae, seizure	severe pht	5b	yes	no	no	no	3	9	improved	no
pachaiappan	66	male	nil	no	5b	no	yes	yes	no	4	5	improved	no
balu	54	male	cad	no	4	no	yes	no	no	1	4	improved	no
banu	55	female	hypersenstivity pneumonitis	mild PHT	5b	no	yes	yes	no	4	17	improved	no
natarajan	54	male	nil	no	5b	no	yes	yes	no	4	2	mortality	yes
jeyaraman	65	male	PT SEQUALAE	no	4	no	no	no	no	0	5	improved	no
vellammal	55	female	nil	no	5b	no	no	no	no	2	8	improved	no
narayanan	63	male	nil	no	5a	no	no	no	no	1	8	improved	no
soundiah	72	male	PTB	no	4	no	yes	no	no	1	3	improved	no
kumar	47	male	CAD	severe pht	4	no	no	no	no	0	5	improved	no
chandrahasn	68	male	nil	no	4	no	no	no	no	0	4	improved	no
muniandi	68	male	nil	no	4	no	no	no	no	0	3	improved	no
akbar ali	67	male	nil	no	4	no	no	no	no	0	5	improved	no

feroz khan	61	male	nil	no	4	no	no	no	no	0	4	improved	no
dayalan	55	male	nil	no	4	no	no	no	no	0	4	improved	no
rajendran	47	male	nil	no	4	no	no	no	no	0	5	improved	no
thangavel	52	male	nil	no	4	no	no	no	no	0	4	improved	no
radhakrishnan	65	male	PT SEQUALAE	no	5a	no	yes	no	no	2	5	improved	no
yamunapathi	60	male	pt sequalae,	no	4	no	no	no	no	0	4	improved	no
chellapa reddy	60	male	nil	no	5b	no	no	no	no	2	18	improved	yes
Duraisamy	72	male	hypersenstivity pneumonitis	severe pht	5b	no	no	yes	no	3	7	improved	no
karuppiah	61	male	nil	no	4	no	no	no	no	0	4	improved	no
padmanaban	82	male	nil	no	4	no	no	no	no	0	4	improved	no
samikannu	47	male	nil	no	4	no	no	no	no	0	5	improved	no
jeganathan	57	male	nil	no	4	no	no	no	no	0	4	improved	no
munusamy	52	male	nil	no	4	no	no	no	no	0	4	improved	no
thiruselvam	74	male	connective tissue disorder	no	4	no	no	no	no	0	3	improved	no
thulasi	45	female	nil	no	4	no	no	no	no	0	4	improved	no
sugumar	46	male	PT SEQUALAE	severe pht	5b	no	yes	yes	no	4	13	improved	yes
anbuchelian	68	male	PT sequale	no	4	no	no	no	no	0	4	improved	no
kasi	48	male	nil	no	4	no	no	no	no	0	4	improved	no
krishnasamy	74	male	pt sequalae	no	4	no	no	no	no	0	3	improved	no
velu	53	male	nil	no	4	no	no	no	no	0	3	improved	no
nathan	56	male	nil	no	4	no	no	no	no	0	5	improved	no
elancheralathan	68	male	nil	no	4	no	no	no	no	0	4	improved	no
ignasi muthu	57	male	nil	no	4	no	no	no	no	0	4	improved	
kuppan	75	male	nil	no	4	no	no	no	no	0	3	improved	no
nallakannu	67	male	nil	no	4	no	no	no	no	0	3	improved	no